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THE
PRINCIPLES AND PRACTICE
OF
MEDICINE

BY THE LATE

CHARLES HILTON FAGGE, M.D., F.R.C.P.

PHYSICIAN TO, AND LECTURER ON PATHOLOGY AT, GUY'S HOSPITAL; EXAMINER IN MEDICINE IN THE UNIVERSITY OF LONDON

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THE

PRINCIPLES AND PRACTICE OF MEDICINE

VOL. II

DISEASES OF THE HEART AND BLOOD-VESSELS.

FUNCTIONAL AFFECTIONS OF THE HEART

Changes in the frequency of the pulse—Irregular and intermittent pulse—Alternate and twin pulse—Palpitation—Cardialgia—Irritable Heart—Treatment of the above conditions.

ANGINA PECTORIS—*Onset, course—Symptoms—Event—Pathology—Ætiology—Prognosis—Treatment.*

Syncope—Distinction from epilepsy and from sunstroke—Treatment.

IN dealing with the affections of the heart, I shall take first those disorders of which the phenomena are comparatively simple and dependent upon no discoverable organic change, and afterwards those in which more or less marked lesions are present. Under the former head (which is almost, if not quite, coextensive with that of *neuroses* of the heart) I shall have to discuss :

Changes in the frequency of the heart's beats. Arrhythmia and Allorrhythmia, *i.e.* intermission and irregularity of the heart's beats. Palpitation. Cardiac pain and angina pectoris. Fainting or syncope.

CHANGES IN THE FREQUENCY OF THE HEART'S BEATS.—The rate of the heart's contractions is commonly estimated by counting the number of beats of the left ventricle per minute, as indicated by the pulsations of the radial artery at the wrist. And provided that they possess a fair amount of force, are separated by appreciable intervals, and are equal (or at least not very unequal) in character, this answers perfectly well. But sometimes the radial pulse gives no correct information as to the frequency of the heart's action, the stethoscope alone enabling one to count its contractions satisfactorily. This is the case when they are very frequent and feeble, and especially when they differ widely among themselves in force, so that some of them fail to transmit a wave of blood so far as the smaller arteries. With regard to the rate of the pulse it is almost impossible to avoid an ambiguity of expression, which yet is capable of sometimes giving rise to serious misunderstandings. Almost every one speaks of the pulse as being "slow," or as being "quick" or "rapid," when what is meant is that it is infrequent or frequent. In strictness, a *slow* pulse (*pulsus tardus*) is one in which each individual pulsation of the heart takes more than usual time for its completion; a *quick* or *rapid* pulse (*pulsus celer*) is one in which the ventricular contraction is short and soon completed. In the present chapter, at any rate, I shall endeavour to maintain this distinction, employing the terms frequent and infrequent whenever I am referring to the number of beats per minute.

As is well known, the frequency of the pulse is in health liable to wide variations in different circumstances. It is greater in the young than in the old, in women than in men, under exertion than during rest, in the upright posture than in sitting or lying, after a meal than when fasting. It is also liable to be enormously increased by emotion or excitement. There are many persons who cannot place themselves under a medical examination, especially for life insurance, without the heart's beats at once rising to 120, 130, or 140 in the minute. One must always be alive to this source of error, which is, however, often to be avoided by counting the pulse a second time after an interval, when the effect of one's presence, as a stranger, has a little worn off. It is said that the pulse may permanently stand at 100 in a healthy individual; but it is doubtful whether a pulse habitually above 80 ought not to be regarded as evidence of some morbid affection of the heart, at any rate from an insurance point of view; and this was the opinion of Dr Latham ('Works,' Syd. Soc., vol. ii, p. 526).

(1) The diseases in which the frequency of the pulse is *increased* are infinite. They include not only all pyrexial affections, but a large proportion of non-febrile affections of whatever nature, and the great majority of affections of the heart itself.

There are, however, some cases in which a very frequent action of the heart appears to constitute a disease by itself. Three such instances are recorded in the 'Brit. Med. Journ.' for 1867 by Dr Cotton, Sir Thomas Watson, and Dr Edmunds respectively. The patients were all males of middle age. In two of the cases there were several attacks at varying intervals, each lasting from a few hours to two or three weeks. The rate of the pulse was from 200 to 230, yet it was perfectly regular. The termination of the attacks seems always to have been absolutely sudden; in Sir Thomas Watson's patient, on one occasion, the beats of the heart, directly after having been counted at 216, fell to 72, exactly one third of the former number. Dr Edmunds suggested that this fact indicates some analogy between the rhythm of the heart in such cases and the action of a monocord vibrating with two nodal points so as to produce its second harmonic; but the idea appears to be somewhat far fetched. Dr Cotton thought that his patient derived benefit from digitalis. But Dr Walshe states that in the cases of this kind which he has seen that remedy has been useless, nervine tonics being the only medicines that seem to be really of service. His patients were females, but they were by no means all of them hysterical or nervous, some being distinguished by force of character. The causes which he recognised were pedestrian excursions, the ascent of mountains, acute pain with effort to control its manifestation, prolonged mental distress; in not a few instances no cause could be discovered. In Dr Cotton's case the attacks were preceded by gastric disorder. The affection is not always devoid of danger. A sensation of faintness, dyspnœa, and even œdema of the lower limbs have been present in more than one instance; and Sir Thomas Watson's patient died during his fourth seizure, the heart on post-mortem examination being found large, as if it had been distended, while its muscular walls were very thin and soft.

In 1870 Dr Wilks brought under the notice of the Clinical Society certain cases in which an extreme frequency of the pulse, associated with alarming dyspnœa and with palpitation, was due to nephritis, which itself was generally the result of scarlatina, although in some instances the indications of that disease had been almost unnoticed until the urine was found albuminous. In almost every instance recovery took place within a few days, notwithstanding that the symptoms appeared to be of the most alarming character. In the treatment Dr Wilks recommends purging, cupping, and salines with antimony, rather than the administration of stimulants.

(2) An abnormally *infrequent* pulse is natural to some persons, in whom the heart never beats oftener than fifty or even than forty times in the minute; this does not, so far as I am aware, affect either the prospect of longevity or the capability of recovering from illness. Among affections which may render the pulse infrequent I may mention aortic stenosis, fatty degeneration of the heart (in some cases), and jaundice; this condition is also observed during convalescence from various acute diseases. I do not know that it ever occurs by itself as the sole or principal indication of disturbance of the patient's health.

ARRHYTHMIA.—*Intermittence and irregularity of pulse.*—One of the more common kinds of perversion of the cardiac rhythm is that in which the heart from time to time "intermits" or leaves out one of its beats, or even two or three successive beats, its action in all other respects being perhaps perfectly regular. This, which might appear to be an alarming occurrence, is rather to be regarded as "the slightest form of derangement of the action" of the organ, to use the words of Dr Balfour. Sometimes the intermissions take place at intervals of only a few pulsations, sometimes not oftener than once in two or three minutes; sometimes they are themselves regular, sometimes altogether irregular in the frequency of their recurrence. The patient may be quite unconscious of any disturbance of the cardiac function, or he may (according to Dr Walshe) experience an instantaneous and transitory feeling of faintness. Much more often, I think, what draws his attention to the fact that his heart now and then misses a beat is that the beat which follows every intermission has an unnaturally thumping character. One may then say that besides the intermittence he suffers from palpitation. Even apart from subjective sensations it may sometimes be noticed that there is an unusual force and fulness of the pulse in the radial artery after each pause in the heart's action. And in some cases the beats of the heart are perpetually varying among themselves not only in their character but also in the rate of their repetition. Another feature may be that the organ occasionally makes a series of short ineffectual contractions in rapid succession, causing what the patient describes as a fluttering sensation within the chest. As Dr Balfour remarks, this may occur only at very long intervals, perhaps not oftener than once in several months. A curious point, of which I have had personal experience, and which has been confirmed by the statement of at least one of my patients, is, that when a person is accustomed to having his attention drawn to simple intermission of the pulse by slight feelings of palpitation in the left side of the chest, similar feelings may be occasionally experienced without any intermission taking place. I have often fancied, under such circumstances, that there was a momentary contraction of fibres of an intercostal muscle, in obedience to some association of the superficial and deeper structures analogous to that pointed out by Van der Kolk and by Hilton.

Even the combination of extreme irregularity with frequent intermittence of the heart's action is sometimes a much less serious matter than might have been expected, especially in persons no longer young, who are able to lead quiet lives, and are neither called upon for active exertion nor compelled to endure the pressure of emotion. Such persons often continue to live for years, and pass their days happily and even usefully to others. This kind of cardiac disorder, however, may be a symptom of dangerous organic disease; even when no murmur can be detected with the stethoscope, there is always the possibility that some degenerative change in the walls of the left ventricle may be present.

As a rule, I think that irregularity of the pulse should be regarded as of more importance than mere intermittence. There is, however, one kind of irregularity which signifies nothing, though I have known it made the subject of grave consideration when observed in a candidate for life insurance. I refer to a temporary acceleration of the pulse, for perhaps ten or twenty beats at a time with subsequent slackenings, occurring in nervous persons, while one has one's fingers on the wrist, as the result of transient waves of excitement or uneasiness, produced by the thought of being under medical examination.

But intermittence of the heart's action should never be made light of until the state of the organ itself and that of the vessels have been thoroughly investigated. If there be any undue arterial tension in particular, it should be carefully noted; I can recall one well-marked instance in which intermittence of the pulse, observed from time to time during three or four years, was at the end of that period followed by indications of organic disease of the aorta and of the aortic valves. I suspect that it is always a serious matter if the heart's beats begin to intermit after slight exertion, such as walking a little faster than usual, or hurrying for fifty or a hundred yards to get in time for a train.

Otherwise, there is no doubt whatever that occasional or even habitual intermittence of the pulse is in many persons compatible with a good state of health in all other respects, and with a fair prospect of longevity. Dr Richardson relates that he once made an autopsy in the case of an aged man, in whom for many years the pulse had always intermitted as often as once in eight beats; he found the heart perfectly healthy and the coronary arteries normal.

Dr Walshe remarks, that some people actually feel more comfortable when the rhythm of the heart is irregular than when it becomes (as it sometimes will) perfectly regular. It is also said that the tendency to intermittence passes into abeyance for the time during any illness attended with pyrexia. In one case Dr Richardson ('Trans. St And. Med. Grad. Ass.,' 1869) found the pulse intermittent in an infant on the day of birth, and this condition lasted for five years, after which it gradually disappeared; in another case he found it present in a boy five years old, who afterwards became entirely free from it. In adults, as a rule, it is due to some one of the complex conditions which are included under the term dyspepsia. Dr Balfour, indeed, is disposed to think that it is rarely dependent upon any form of indigestion except that which is associated with a gouty tendency. But in this I cannot agree with him. A circumstance which I have observed with regard to it seems to me to be very suggestive as to its mode of production. As is well known, intermittence of pulse, when the result of dyspepsia, is very apt to come on after the patient lies down in bed. Now, I have noticed in my own person—and patients have assured me that they have noticed the same thing—that when the pulse is intermittent overnight, it often is so on the following morning also, although it becomes regular after one has risen from the recumbent posture. But the process of digestion, if it was going on at bedtime, must have ceased during the night. The probable explanation seems to me to be that which causes the heart's action to intermit is the presence in the stomach of solid pieces of food imperfectly masticated. It is well known that lumps of potato and other substances may remain in the gastric cavity for days together; and nothing seems to me more likely than that when one lies down such masses should fall from the greater curvature of the stomach towards the cardia, and should consequently irritate the terminal branches of the vagi spread over that part of the mucous membrane. In some persons, however, intermittence of the pulse is sure to follow indulgence in tea, especially in green tea; and in others tobacco-smoking has a like effect. Again, Dr Walshe cites a case, recorded by Nooth,* in which a shot, lodged in a bronchial tube, set up asthmatic seizures and rendered the pulse extremely irregular, both symptoms disappearing when it was expectorated. In many persons intermittence of the heart's action appears to be traceable to overwork or to worry rather than to any other cause. Lastly, it sometimes results from the shock of some emotion, such as sudden terror or grief. Dr Richardson and Dr Balfour allude to cases in which it was set up by a railway accident or by a shipwreck; it would seem that the tendency then is for the intermissions to take place gradually at longer intervals until they ultimately cease altogether. Dr Richardson relates the cases of two patients in whom intermittence of the pulse preceded an attack of mania, in one of them on several different occasions.

Allorrhythmia.—In some cases disturbance of the heart's rhythm shows itself, not in irregularity or intermission, but in the regular succession of beats of different degrees of force, or in the systematic coupling together of beats in pairs. The former variety is named by Traube the *Pulsus alternans*, the latter the *Pulsus bigeminus*. By Sommerbrodt ('Deutsch. Arch.,' xix) they are included together under the common name of "allorrhythmia." I believe that I was one of the first to record an example of the *pulsus alternans* ('Guy's Hospital Reports,' xvi, 1871). The patient was a girl, who came under my care for mitral stenosis, attended with a well-marked presystolic murmur. The usual rate of her heart's action was about 70. But sometimes it would rise to 92, and then only every other beat produced a pulse at the wrist, which accordingly was counted at 46; there was, however, reason to believe that the beats which failed to reach the radial arteries was attended with reflux into the systemic veins, inasmuch as a pulse could be felt at the root of the neck, apparently in the jugulars. It is worthy of notice that 70 is almost exactly the arithmetical mean between 46 and 92, for this fact perhaps justifies the conclusion that the efficiency of the alternate beats which were not felt in the arteries was just half that of the regular beats. At one time the allorrhythmic state of the pulse could in my patient be stopped at will, by making her walk so as to quicken the heart's action; the rhythm was then normal, but afterwards, when the heart began to slow again, it fell into the peculiar alternate rhythm. Digitalis, too, seemed at one time to be concerned in producing it, as has been suspected also in some cases of the *pulsus bigeminus* recorded by German observers. An allorrhythmic state of the heart's action, however, is not peculiar to cardiac affections; it has been observed in cases of cerebral hæmorrhage or softening (hitherto only when the right side of the brain was affected?), and also in one case during convalesence from acute peritonitis. How it is produced is not yet clear. Traube, on the ground of some experiments on animals in which he noticed a similar phenomenon, attributed it to the concurrence of two distinct conditions, (1) the withdrawal of the influence of the spinal part of the inhibitory nervous apparatus; (2) the presence in the blood of some substance capable of stimulating the still active cardiac part of the same apparatus. But in one point Traube was entirely wrong, namely, in supposing the *pulsus bigeminus* to be a sign of the near approach of death. This is clear from the cases that have been recorded by different observers; and I may mention that my patient lived ten or eleven years after the publication of her case. Sommerbrodt seems to me to be near the mark when he compares this state of the heart with that modification of the breathing which is associated with the names of Cheyne and Stokes. Just as the latter depends upon rhythmical changes in the degree to which the respiratory centre is stimulated by the blood that circulates in it, so the latter may perhaps be imagined to result from a like influence of the blood supplied to the cardiac ganglia. It is to be noted that the same cases in which the pulse is at times allorrhythmic are apt at other times to present arrhythmia, or (in other words) irregularity and intermittence.

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* 'Trans. of a Society for the Improvement of Medical Knowledge,' vol. iii.

PALPITATION OF THE HEART.—In ordinary circumstances and in a state of health, one is unconscious of the heart's action—if one becomes aware of its pulsations, palpitation is said to be present. This need not necessarily be a morbid affection; it may occur to any person after great exertion, or under strong emotion. But, apart from such causes, it may be due to various kinds of disturbance of the heart; and it sometimes appears to be the chief, or even the only thing that a patient complains of, so as to constitute a disease by itself. In some cases in which the cardiac pulsations seem to the patient to be extremely violent, the physician may find, on placing his hand over the left side of the chest, that they are in reality perfectly quiet and natural in character. Much more often the subjective sensation corresponds with the fact that they are greatly increased in force, they may shake the chest, or the entire body, or even the couch on which the patient lies. Other feelings which distress him may be a violent throbbing of the carotids, a sensation as though "the heart were jumping into the throat" or "the eyes bursting from the sockets," flashes of light before the eyes, dizziness, faintness, or an indescribable sense of discomfort in the region of the heart, rarely amounting to actual pain. The attempt to lie on the left side often greatly aggravates the symptoms. There is often extreme anguish with a fear of impending death. On examination the apex beat is seen to be in its natural position, but it occupies too extensive an area. Dr Walshe describes the impulse as feeling like a *blow,* with even somewhat of a heaving character, if the heart is well nourished; like a *slap,* if the organ is feeble. The area of percussion dulness is usually normal in extent, but in prolonged paroxysms there may be some increase of it to the right of the sternum. The sounds are loud and clear, with a metallic, ringing character; the first sound can sometimes be heard at a distance of some inches from the chest. Dr Walshe speaks of a basic systolic murmur as of frequent occurrence in patients who are in the slightest degree anæmic. He is also disposed to suspect that a transitory condition of mitral regurgitation may be present, causing a systolic murmur at the apex; I should myself be inclined to scrutinise very carefully the relations of any such murmur to the respiratory rhythm, for cardiac palpitation seems extremely likely to give rise to a peculiar form of murmur which will be described elsewhere as resulting from disturbance of the edge of the left lung by the heart's movement. The pulse is not necessarily increased in frequency; Dr Walshe says that in vigorous and plethoric subjects its rate may be normal or even below normal.

Palpitation of the heart is commonly present in various forms of organic cardiac disease, and the physical signs are then of course modified by those which belong to the particular affection that may be present. It may also accompany other forms of functional or neurotic disturbance of the organ; in exophthalmic goitre it is one of the principal symptoms. In these circumstances it may be more or less persistent. On the other hand, when it occurs by itself, it is usually paroxysmal. The duration of an attack may then be from a few minutes to several hours. Dr Walshe remarks that it may terminate with an abundant flow of watery urine, and that as it subsides the patient often falls asleep.

It is especially during early adult life that palpitation is apt to occur as a substantive disease. Nervous hysterical females are very liable to it; but the most severe cases of all are seen in excitable males. An important part in its ætiology seems to be played by exhaustion from over-study, sexual excess, masturbation, the abuse of alcoholic stimulants or of tobacco, indulgence in strong tea. But in some cases none of these causes can be made out. I believe that it is then very often due to overloading of the stomach with imperfectly masticated and indigestible food, especially shortly before bedtime. It has appeared to me that, like intermittence of the pulse, palpitation may have such an origin, even when there is no sense of fulness or discomfort at the epigastrium, nor any other of the common symptoms of dyspepsia. It is especially when the affection recurs night after night when the patient is in bed that this cause should be suspected. In other cases it comes on during exertion, as when the patient begins to walk, even slowly and on the level. Dr Balfour says that one characteristic of the nervous or functional nature of the palpitation in such circumstances is that it disappears if he exerts himself a little more, as by taking a short run. In other instances palpitation may be noticed to subside under any emotional excitement of a pleasurable kind.

Cardiac Pain.—Pain is a frequent, though not a constant, symptom of many affections of the heart, functional and organic; on the whole, perhaps it is more marked in the former than in the latter. Its most usual seat is a localised spot a little outside the left nipple, beneath the fourth intercostal space, or the fifth rib; but it may be situated near the sternum, or rather to the right of it. Dr Walshe says that it is evidently deep seated, and not in the cutaneous nerves; but as to the correctness of this distinction I must confess to a doubt, which I have discussed under the subject of neuralgia (vol. i, p. 358). It is generally more or less paroxysmal in character and is often described as sharp or lancinating, sometimes as burning, tearing, or cutting. There may also be a constant dull heavy pain, and in some cases this exists by itself. It is often increased by exertion or fatigue, and it generally undergoes aggravation when palpitation occurs. It often radiates widely over the side of the chest, up into the neck, into the axilla and down the left arm, even sometimes down the right arm. When it thus affects the arm it is apt to be associated with a sensation of numbness or tingling; it sometimes ceases abruptly at the inner side of the elbow, sometimes extends down the forearm to the inner side of the hand. In some cases its principal seat is behind, near the angle of the left scapula. Dr Walshe says that tenderness of the surface is absent; pressure upon the principal spot rather relieves than increases the pain. Da Costa, however, in describing some cases to which I shall refer presently, says that the cardiac region was hyperæsthetic, especially after attacks of palpitation.

A modification of cardiac pain described by Dr Walshe, is one which comes on when the patient bends forward, as in pulling on his boots. It is relieved by stretching out the chest wall and pressing on the surface. Dr Walshe says that he has observed it more often in elderly than in young persons. He thinks that it may depend upon "twisting of the præcardial costal cartilages, which have lost the flexibility of youth," but I should have been rather disposed to attribute it to upward pressure upon the heart through the diaphragm. I am not sure whether it ought not to be regarded as merely a form of angina pectoris; at any rate, it is not seldom present in that disease, as is shown by the cases recently published by Dr Murrell in illustration of the treatment by nitro-glycerine.

Irritable heart.—The close connexion between the various forms of functional or neurotic disturbance of the heart is well illustrated in a very interesting paper by Da Costa ('Am. Jour. of Med. Sc.,' 1871) on what he terms "irritable heart." This paper is further of great importance as containing what is almost complete proof of the gradual passage of such an affection into a condition of organic cardiac disease, the organ gradually undergoing hypertrophy. It is based upon no fewer than three hundred cases of soldiers in the Army of the United States during the Civil War. The general history of these men is that having been called from civil pursuits into active service without previous training, they became liable to attacks of palpitation, to more or less severe pain in the chest, of a sharp and stabbing, or of a dull aching character, and to dyspnœa on exertion, so that they became unable to keep up with their comrades and were distressed by the weight of their accoutrements. On examination the pulse was found to be much increased in frequency; it was much influenced by position, there being sometimes a difference of thirty beats or more between its rate during standing and that during lying down; in some instances it was intermittent or irregular. Yet the men themselves often looked strong and healthy, though their hands were apt to be bluish and mottled, and to be easily made pale by pressure. The cause of the affection was sometimes merely hard service in the field, particularly excessive marching. But in many cases it was directly brought on by an attack of diarrhœa, not sufficient to interfere with duty; or by fever, necessitating a few days' stay in hospital. The patients were generally young men, from sixteen to twenty-five. The course of this affection was usually slow. But after some months of treatment it often ended in complete recovery, so that active exercise of all kinds could be borne without discomfort. In other cases the cure was imperfect, there being still a liability to cardiac symptoms on exertion. Hypertrophy of the heart was believed to have developed itself in twenty-eight cases out of two hundred.

Da Costa's observations with regard to the *treatment* of his cases are of great interest and importance. Rest was found to be very useful. Making the patient lie down for several hours daily often led to strikingly good results; and two men who were kept in bed—one by an attack of dysentery, the other by a broken leg—improved very rapidly. Among ordinary civil patients, in whom we have seen that functional disorder of the heart is often due to overloading of the stomach, regulation of the diet is a very essential part of the treatment. Great moderation in the use of stimulants, or even total abstinence, should be insisted on, in most cases. Care must be taken that neither tobacco nor tea is used to excess; all forms of excitement, including sexual indulgence, must be kept under control. When there is anæmia, tonics such as iron, zinc, or quinine, may be prescribed with advantage.

General treatment.—Among drugs intended to affect the heart directly, digitalis is by far the most useful. This was Da Costa's experience, and I think that it is in entire accordance with that of other observers. There were, however, a few cases in which it had no effect either in modifying the frequency of the pulse, or in correcting its irregularity. In cases of simple palpitation, such as are described at p. 6, one might have doubted, on theoretical grounds, whether digitalis would do good. Dr Walshe, however, says that clinical experience is in its favour. Other medicines which may be of service are the bromide of potassium or of ammonium, hyoscyamus, cannabis indica, ammonia, ether (especially the spirit. ætheris comp.); and in hysterical females, assafetida, musk, or valerian. Schrötter recommends the application to the cardiac region of cloths wrung out in cold water, or even of an icebag; but Dr Walshe says that this is a dangerous practice, especially if the rhythm of the organ be in the smallest degree affected.

For palpitation accompanied by great irregularity and intermittence of the pulse, it is agreed by all writers that alcohol is one of the best remedies. Dr Walshe says that from a teaspoonful to half a wineglassful of brandy should be given, according to circumstances. It is, however, very important to take care that dram-drinking shall not become a habit. Digitalis is often of very great use in steadying the heart's action, and Da Costa found that belladonna or atropine was especially likely to do good in cases in which the pulse intermitted.

Aconite seems to be of little or no service in cases of mere functional palpitation, but Da Costa obtained striking results from it when the heart was beginning to undergo hypertrophy. It often exerted a marked influence upon the force of the cardiac beats, without diminishing their frequency, whereas exactly the opposite effect was produced by digitalis. Consequently, in suitable cases the two drugs were given together with great advantage. Veratrum viride seemed to be intermediate in its action between them.

A point on which Da Costa laid great stress in the management of his patients, and which is doubtless of no less importance in like cases occurring in civil practice, was the maintenance of great care during convalescence. Before allowing the men to return to their regiments, he tested them by running and by other exercises, so as to see how the heart bore itself under strain. He gives reports of some cases which came under notice again after an interval of several years, and in which no relapse had occurred.

Angina Pectoris.—Under this name—sometimes rendered into English as "breast-pang"—is known a very severe form of cardiac pain, occurring in sudden short paroxysms, and accompanied by a sense of impending death, which is not seldom actually realised. It was first described in 1768 by Heberden, who had observed several cases of it. French writers have recently set up a claim of priority for one of their countrymen, Rougnon, on account of a letter written by him to Lorry a few months earlier, in which is related the death of a cavalry officer, M. Charles, by what was probably the same disease. But Dr Gairdner points out ('Reynolds' System,' iv, p. 537) that in so far as the account of a single case can be held to have anticipated Heberden's observations, the merit really belongs to Morgagni, who recorded a similar instance in a Venetian woman early in the eighteenth century. In its well-marked and typical form angina pectoris is rare. Cases are not infrequent, which more or less closely resemble it in the character of the pain, although in these there is not the same danger to the patient's life. Whether or not these should be classed under the same heading is a question that can be satisfactorily discussed only after the pathology of the affection has been considered. At present I shall confine my description to the classical examples of it.

Onset and exciting causes.—Angina pectoris is almost always absolutely sudden. The first attack in most cases occurs while the patient is walking, especially on rising ground, or with a strong wind against him, or shortly after a meal (Dr Walshe says, most often after the earliest meal in the day). But sometimes (as in the case of Arnold, of Rugby, recorded by Dr Latham) a person who has never before suffered from it is awakened by it from his sleep. Subsequent seizures are apt to be brought on by comparatively slight causes. Thus the patient may gradually find that one form of exertion after another is unsafe for him. Or emotional excitement may give rise to it, as in the well-known case of John Hunter, who died within the walls of St George's Hospital in consequence of a dispute at a meeting there, with regard to which he had previously expressed a foreboding that the result would be fatal to him. Dr Walshe has even seen a paroxysm brought on by emotion of a pleasurable character. In many instances the seizures, which at first took place only during the daytime, at length begin to recur even in the night. Dr Latham alludes to one patient who was attacked as soon as he lay down. The act of stooping, as to pull on the boots, or even in washing, is a frequent cause of the attacks of angina. In some cases they are brought on by such slight efforts as coughing, defæcation, or the hasty swallowing of cold water.

Symptoms.—The chief seat of the pain in angina pectoris is usually behind the lower part of the sternum, rather to the left than the right side; sometimes behind the middle or the upper part of that bone. When the patient can attempt to describe it, he generally speaks of it as gnawing, tearing, or lancinating in character. But in many cases it seems to be altogether indescribable—a torture so intense that he feels that any increase of it, or even its continuance, must bring his life to an end. The pain often spreads round, generally through the left side of the chest, to the spine. It is sometimes accompanied by a sense of constriction, as though the sternum were forcibly drawn backwards: Dr Gairdner cites the case of a medical man in whom there was a subjective sensation as though the front of the chest were "bulged out in a convex prominence, terminating suddenly at the lower end of the sternum in a sharp and deep depression." Very often the pain radiates upwards into the neck or towards the occiput, down the left arm to the elbow or into the fingers, occasionally down the right arm, into the lower limbs or into the testes. In these distant parts it may be accompanied by feelings of tingling or of numbness. In some cases, as in one observed by Dr Walshe, it takes a course the reverse of what is usual, beginning at the left wrist and extending upwards to the heart. In the præcordial region there is often tenderness to pressure; but sometimes friction gives relief.

A patient attacked by this affection is instantly arrested in whatever he may be doing; if walking he is motionless until the seizure passes off; if standing, he dares not sit down. It is, however, a curious circumstance that some persons, after having been pulled up by the pain three or four times at the beginning of a walk, will afterwards go on with ease for several miles. The feeling of constriction in the chest may cause him to speak of experiencing a "want of breath" or a "sense of suffocation;" but all observers are agreed that there is never any dyspnœa in the proper sense of that term, and that no lividity of the features shows itself. The breathing may indeed be somewhat increased in frequency, but this is because the patient instinctively keeps the thoracic movements as shallow as possible, for fear of increasing the pain. By an effort of the will he can, if he chooses, freely expand the chest. There are even exceptional cases in which drawing a deep breath gives momentary relief to his suffering.

With regard to the state of the heart's action and of the pulse during a paroxysm of angina pectoris, the accounts of different writers have differed widely; and I think that what we shall presently find to be the probable pathology of the disease favours the conclusion that all cases are not alike in this respect. It is said that the impulse and the sounds of the heart are sometimes unaltered in character throughout the seizure, and the pulse regular and neither frequent nor weak. Dr Walshe, speaking from his own experience, declares that at least towards the close of a paroxysm, when the patient is about to recover, there may be no acceleration nor any irregularity of beat. But Dr Parry long ago described the pulse as being more or less feeble, according to the violence of the attack, and Dr Gairdner expresses the same opinion. In many cases it is expressly noted that the pulse has been small, irregular in rhythm, but not necessarily increased in frequency, and sometimes morbidly infrequent. The failure in the circulation is also shown by deadly pallor of the face, by coldness of the limbs, by the presence of clammy perspiration. In cases about to prove fatal, the pulse becomes imperceptible a little while before death.

The mental faculties usually remain unimpaired throughout the seizure; but after its subsidence the patient is said sometimes to have no remembrance of anything except the intense agony which he has undergone. Thus the state of the brain in angina pectoris would appear to resemble that which is present during collapse, and not that during syncope. But Dr Walshe says that the sight sometimes fails more or less completely. There may be slight convulsions, or even tonic spasms of a very severe kind, with opisthotonos.

There is in some cases violent and continued eructation, or vomiting; or the whole abdomen may become distended with flatulence. A copious flow of watery urine sometimes occurs as the attack is passing off. Trousseau relates a case in which the paroxysms, though frequently repeated, and though each lasted only about a minute, were always accompanied by an irresistible desire to micturate.

The duration of an attack of angina pectoris is usually a few seconds or minutes; but attacks may recur again and again for an hour or longer. A gentleman to whom I was called in the north of London remained for many hours stooping over the end of a couch, refusing to move for fear of the return of the pain. But sometimes when a seizure occurs during walking, it ceases as soon as the patient stands still. Trousseau remarks that very different attitudes are assumed in different cases. One patient will be motionless on his back, another will incline backwards on his chair or on pillows, a third may place himself on all fours, resting on his knees and his elbows, a fourth may bend forwards as far as possible.

Fatal event.—When this disease proves fatal, the heart is found at the autopsy to be relaxed and flabby, even though there is marked cadaveric rigidity of the muscles generally. Dr Walshe says that there is an almost complete absence of blood from the cardiac cavities, which fact certainly looks as though a ventricular systole not followed by an active dilatation had been the last act of life. Sometimes death appears to be absolutely instantaneous; Dr Walshe relates an instance in which the patient had been reading quietly in bed, and in which the thumb and the forefinger were found in the pamphlet on which he had been engaged, the bedclothes being also quite undisturbed. In some of these cases even where there have been former attacks attended with severe pain, it seems probable that the fatal seizure must have been so brief as to be painless. And one may fairly suspect that sudden death, in persons who have never been known to suffer from angina, is not seldom essentially of the same nature. But sometimes death is more gradual, being preceded by gradual failure of the pulse, laboured breathing, and unconsciousness. We have seen that, as a rule, the paroxysms of angina pectoris return again and again; there is generally an interval of some years between the first attack of the disease and its fatal termination. Whether a single paroxysm ever occurs without being followed by any others, seems to be doubtful, though it is certain that the patient himself may do a great deal to prevent their recurrence by avoiding exertion and emotion. Dr Walshe speaks of having himself seen a patient who appeared to have been first attacked twenty-four years previously; and there is some reason to believe that John Hunter had begun to have seizures of angina twenty years before his death. But the longest case that I know of is one related by Dr Murrell, in which the patient had suffered for thirty years, the diagnosis of angina pectoris having been formally given by Sir Risdon Bennett twenty-six years before he came under Dr Murrell's notice. In some instances the disease returns very frequently, so that the whole number of paroxysms must be very large. On the other hand, Dr Walshe relates an instance in which there were only three, one being a year, the other half an hour, before the third, which proved instantaneously fatal. Dr Latham met with two cases in one of which death occurred fourteen days, in the other ten days, after the first attack. The most rapid case on record is, I believe, that of Arnold of Rugby, who, having never suffered from angina, went to bed on the 11th of June, 1842, in apparent health, but was first seized with the pain at about five a.m. and died soon after a quarter past seven.

Pathology.—The pathology of angina pectoris is still obscure. Trousseau and Anstie have maintained that it should be regarded as a neurosis, or a "visceral neuralgia," and the same opinion is formally upheld by Eulenberg in Ziemssen's 'Handbuch,' though perhaps a more correct statement would be that he rejects it, since he would exclude from the disease, in its "purely nervous" form, the very cases involving danger to life, on which the foregoing description is based, and which from the days of Heberden to the present time have been regarded as the true and typical examples of it. For a neuralgia to prove habitually fatal is, indeed, without precedent, and another point in which angina pectoris differs from all neuroses is in being generally, if not always, associated with the existence of organic lesions in the heart or in the great vessels, although it would seem that no one lesion is constantly present. This, at any rate, is true of the cases that destroy life, and that occur in middle-aged or old persons, or sometimes in young adults, as in a patient of Dr Balfour's, to whose account I shall refer when I discuss the morbid changes that are found in the disease. Lastly, it is unlike a neuralgia to show, as angina does, an enormous preponderance of males over females—as many as ten of the former to one of the latter—among those who are attacked by it. Anstie, on the other hand, declares that those who suffer from the disease are always of nervous temperament, this being shown by the frequent occurrence of other neuroses in different members of their families, and he also, like Trousseau, insists on the existence of a relationship between angina pectoris and asthma.

Within the last few years observations have been made which tend to show that angina pectoris may depend upon organic lesions affecting the cardiac nerves and ganglia. The earliest record of the occurrence of such lesions, indeed, dates back to 1841, when Heine ('Müller's Archiv,' 1841) published a case treated by Skoda, in which Rokitansky made the autopsy and found the right phrenic nerve, the *N. cardiacus magnus* and the descending branches of the left vagus each involved in pigmented nodules, doubtless altered lymph-glands. The symptoms during life, however, consisted, not in paroxysms of angina, but in attacks of intermittency of the heart's action, continued during a period of from four to six beats, and attended with a feeling of inexpressible anxiety. In 1864 Lancereaux ('Gaz. Méd.,' 1864) had an opportunity of examining the body of a man who had long suffered from angina pectoris and who had at last died suddenly; he found a raised patch in the aorta between the orifices of the coronary arteries, with injection and thickening of the corresponding part of the external coat of the vessel; in this injection the adjacent cardiac plexus took part, and some of its fibres were surrounded by a nucleated material, and presented a greyish, finely granular appearance of their myelin. Similar changes in the nerves are recorded and figured by Peter ('Traité des Maladies du Cœur,' 1883) as having been observed by him in two cases which came under his care, and a fifth instance fell under the notice of Bazy ('Bull. de la Soc. Clin.,' 1878).

Since the year 1867, however, clinical experience has taught in a very decided manner that the immediate exciting cause of the paroxysms of angina pectoris is a sudden rise in the tension of the systemic arteries. A similar view had, I believe, been previously suggested by Traube. But what first proved its correctness was a series of observations made by Dr Lauder Brunton on a patient of Dr Bennett's in the Royal Infirmary of Edinburgh. The man was affected with aortic regurgitant disease, and was liable to frequent attacks of angina-like pain. Dr Brunton found that during these attacks the sphygmographic tracing of the pulse became rapidly altered, the curve being lower, the dicrotism disappearing, and both the ascent and the descent being more gradual—changes indicative of a marked increase of arterial tension. These observations led Dr Brunton to propose the inhalation of nitrite of amyl as being likely to relieve the pain, and the trial of this agent was attended with brilliant success. Subsequent experience has shown that we have in nitrite of amyl and in nitro-glycerine (of which the physiological action is analogous) most valuable remedies for angina pectoris. And although I am not aware that the increase of arterial tension has been since shown by the sphygmograph to be a constant feature of the paroxysms, this fact is perhaps sufficiently established by the therapeutical results which are so frequently obtained. The pallor and the coldness of the face and of the limbs that accompany severe seizures can hardly be cited as evidence in themselves of arterial spasm, for failure of the heart's action might produce the same effects. Trousseau, however, draws attention to the fact that the pallor is sometimes followed by a reddish or livid-bluish hue, and the same thing was noticed in one case by Anstie ('Trans. Clin. Soc.,' vol. iii) and attributed by him to paralytic dilatation succeeding spasm of the arterioles.

Under the name of "Angina Pectoris vasomotoria" Nothnagel has recorded ('Deutsch. Archiv,' iii) a series of cases which seem to have an important bearing upon this question. Their peculiarity lies in the fact that the earliest and most conspicuous symptoms of the paroxysms from which the patients suffered were coldness and pallor with numbness and stiffness of the limbs; the palpitation, the feeling of oppression at the chest, the giddiness, the sense of impending death being all apparently secondary and attributable to the increased efforts which the heart was called upon to make to overcome the resistance opposed to it. The attacks were also definitely traceable to external cold, and were relieved by hot foot-baths and frictions; in fact, the state of the peripheral circulation seems to have been very similar to that which in other patients leads to paroxysmal hæmoglobinuria. Pain appears to have been a much less marked symptom than in ordinary angina; however, a dull pain is mentioned, seated chiefly in the cardiac region, but in one case extending over the whole of the left side of the chest, and sometimes down the left arm.

On the whole, I think that one may take Nothnagel's cases as proving that a sudden increase of tension in the peripheral arteries due to a cause acting upon the body from without is capable, in some persons, of giving rise to phenomena approaching those of a paroxysm of angina pectoris. The next question is how we are to suppose the arterial spasm to be brought about in the more usual form of the disease. Now, if the anatomical researches of Lancereaux and Peter are to be credited with the significance which these observers attach to them, nothing seems to be more simple than to suppose that from the cardiac nerves and ganglia there is transmitted to the vaso-motor centre an impression which causes it to throw the muscular walls of the smaller vessels throughout the body into contraction. But it is, I think, necessary to exercise a good deal of caution before we adopt such a view. The lesions discovered by the French pathologists were after all in every instance secondary. If the nerves lying adjacent to areas of chronic thickening and induration at the base of the heart (or, indeed, elsewhere throughout the body) were to be systematically dissected, is it not probable that they might very often be found involved in the morbid process, even when no symptoms had existed that could possibly be traced to them? It seems to me rather significant that, like Rokitansky, Peter found the phrenic nerves affected in exactly the same way as the cardiac nerves.

One point in which the phenomena of the paroxysm of angina pectoris seem to differ from what might be theoretically expected on the view that it is essentially dependent upon an increased tension in the peripheral arteries is in its not being invariably, or even generally, attended with a reduction in the frequency of the pulse; among Nothnagel's cases, too, there is only one in which a fall from 80 to 64 or 60 beats in the minute is noted.

It must, however, be remembered that in the severe and dangerous form of angina pectoris, which is regarded as typical of the disease, organic changes in the heart and large vessels are usually, if not always, present. Among recent writers Anstie, I think, stands almost alone in maintaining that fatal seizures may occur in persons in whom no such disease exists. As a rule, the most conspicuous lesion is either a soft, flabby, or fatty state of the cardiac muscle, or else a chronic inflammatory change in the coats of the aorta at or near its origin, leading to atheroma, to calcification, and perhaps to aneurysm: Dr Gairdner has specially insisted upon the frequency with which symptoms of angina accompany even small aneurysms, arising very near the heart and projecting into the pericardium. It has, however, long been a question whether one ought not to regard, as more especially connected with angina pectoris, another lesion which is undoubtedly often found associated both with fatty heart and with *arteritis deformans* of the aorta, namely, obstruction of the coronary arteries. This was first suggested by Jenner, the discoverer of vaccination, in a letter which he addressed to Heberden in 1778, but which he did not send out of consideration for the feelings of his friend John Hunter, whom he rightly believed to be at that time a sufferer from angina. Sometimes the coronary arteries have their orifices more or less completely obliterated by disease of the aorta itself, their coats in the rest of their course being healthy; sometimes they are converted into thick calcified tubes in nearly their whole length.

But it would be a position altogether untenable if one were to maintain that obstruction of the coronary arteries is constantly present in cases of fatal angina, either with or without other more conspicuous lesions of the heart or of the aorta. And it is no less certain that these vessels are often found in the post-mortem room to be very greatly narrowed when there were no symptoms of angina during life. Again, the long duration of the disease in some cases seems to be inconsistent with the idea that any of the organic lesions above described can have existed throughout its whole course. Perhaps after all it may be that the paroxysms of angina owe to the lesions in question their severity and their tendency to prove fatal, but do not stand to them in the direct relation of effect to cause. For, if the disease be regarded as a struggle on the part of the heart to overcome an excessive resistance in the arteries, enfeeblement of the cardiac muscle, whether as the result of fatty change or of a mere deficiency of blood-supply, cannot but add greatly to the violence of the seizures. From this point of view it seems probable that many instances of the affection which Dr Walshe describes as "pseudo-angina," and which is admitted to be of far more frequent occurrence, are fundamentally of the same nature; but it would still doubtless be necessary to exclude cases dependent upon hysteria or upon flatulent distension of the stomach. Within a single year I saw two young clerks in the same London bank, each of whom described attacks that could not, I think, have been distinguished from those of angina pectoris, although the ages of the patients rendered it very unlikely that the affection would prove dangerous to life. But that this does not always form a safe criterion is well shown by a case of Dr Balfour's, that of a man, aged twenty-four, who died after four months' illness with paroxysmal pain in the epigastrium, and in whom (as had been correctly diagnosed during life) the base of the aorta presented a ring of atheromatous thickening, by which the mouths of the two coronary arteries were greatly narrowed. Such a case, however, is altogether exceptional. Dr Walshe says that angina pectoris (exclusive of his pseudo-angina) is rare before the fiftieth, excessively so before the fortieth year. In consideration of the view that the disease depends upon an increase of arterial tension, it is worth notice that Dr Walshe speaks of it as never occurring in association with mitral disease, whether in the form of stenosis or of regurgitation; for in such cases the tension in the arteries is, of course, lowered. But I do not think that many observers will agree with him in saying that it rarely accompanies aortic insufficiency, at any rate where this is the result of an *arteritis deformans,* involving the sigmoid valves.

Ætiology.—With regard to the remote causes of angina pectoris, as distinguished from the exciting causes of the paroxysms, I have little to add to what has already been incidentally mentioned. There has been some discussion whether the disease is related to gout; probably if such a connection exists, it is indirect and through the medium of arteritis deformans of the base of the aorta, which is of frequent occurrence in gouty subjects. It is, however, a remarkable circumstance, which was first pointed out by Blane, that angina is much more frequent among well-to-do persons, than among the poor. Dr Gairdner throws some doubt upon this, but I think that there can be little question about it; Dr Walshe says that his experience scarcely supplies him with more than a solitary well-defined example of the affection in hospital practice. In some cases a tendency to it has appeared to be transmitted by inheritance. Dr Gairdner remarks upon its greater frequency in persons who are corpulent and of sedentary habits. It is, however, to be observed that the liability to attacks of angina interferes greatly with the activity of those who had before been accustomed to take exercise.

A point worthy of notice is that persons suffering under frequent attacks of angina pectoris often have a haggard, frightened aspect; this is in many cases due to their sleep being broken by bad dreams; some patients are actually afraid to lie down in bed for fear of the occurrence of a seizure.

Prognosis.—It is important to remember that the cardiac affections most frequently associated with angina pectoris are such as it is at present impossible to diagnose with certainty. Consequently, one must never be led to give a favorable prognosis by the fact that on examination of the chest one has failed to detect evidence of organic disease. Dr Walshe, however, states that in every one of twenty-four cases which had been examined by him during life he was able to make out physical signs of some morbid change either in the heart, or in the aorta, or in both. And Dr Balfour says that he has never met with an instance of angina in which signs of dilatation of the heart were not present. Dr Latham's experience, again, is very similar; among thirteen cases there were only three in which neither increased dulness on percussion nor any murmur on auscultation over the heart or the aorta was present; and even in those three cases the cardiac impulse was most feeble, while the sounds, though "natural in kind," were "raised to their highest intonation and diffused over the entire front of the chest." In the next chapter, however, I shall have to discuss how far signs of dilatation and of other changes in the muscular walls of the heart can be relied on.

But besides being unable to assert, in any case of angina pectoris occurring at a time of life when organic changes in the cardiac muscle are apt to take place, that the disease is free from danger to life, one is also never justified in giving an opinion as to its probable duration. A patient's first seizure may have been mild, yet after a longer or shorter interval it may be followed by another of such severity as to prove fatal. Or whereas the first may have been very severe, the subsequent ones may be far less so.

Treatment.—In the treatment of angina pectoris a great deal more can be done than formerly. The older physicians could recommend nothing better than large doses of laudanum and brandy. But we now, knowing how slowly absorption from the stomach takes place, always prefer to give morphia subcutaneously. And Dr Balfour seems to have shown that it is perfectly safe to use chloroform freely, so as completely to narcotise the patient. When the pain is very severe, he says that this is the only method by which it can be relieved; in protracted paroxysms, he follows it at once by injection of half a drachm of Squire's solution of the bimeconate of morphia beneath the skin of each arm, so that the chloroform sleep may pass into the morphia sleep, from which the patient awakes after some hours, free from suffering but exhausted, and generally with some œdema of the lungs.

But, as a rule, one should rather have recourse to the nitrite of amyl or to nitro-glycerine. The advantage of the former agent is the rapidity of its action. The best way is to employ the glass capsules each of which contains from three to five minims of it. One of them is broken within the folds of a handkerchief, and the vapour is inhaled as freely as possible. In from fifteen to twenty seconds the face flushes, a sense of fulness in the head is experienced, the pulse at the wrist loses its tension, and the pain ceases. Dr Balfour has found in two cases that nitrite of amyl kept in a stoppered bottle gradually loses much of its efficacy in cutting short the paroxysms of angina, notwithstanding that it still flushes the face. I am not aware that clinical experience has as yet confirmed Anstie's apprehension that this remedy might possibly induce cerebral hæmorrhage if the arteries of the brain should be diseased.

Since the year 1877, nitro-glycerine has been largely used in the treatment of angina pectoris; it was first tried by Dr Murrell. Although its physiological action is less rapid than that of nitrite of amyl it is yet commonly quick enough to cut short an attack if taken at the very commencement. But for this purpose it must be employed either in solution or in the form of tablets made with chocolate (by Martindale). Pills, unless broken up by mastication, are too slow in their effects. The proportion most commonly used is an alcoholic solution of the strength of 1 per cent. It may be given in a drachm of water, when it is almost tasteless; or if there be flatulence, in peppermint water with a little chloric ether. The dose necessary to give relief varies very widely in different patients; sometimes several doses have to be taken in succession at short intervals before the pain can be entirely got rid of. In beginning the treatment, it is generally best to prescribe at first half a minim or one minim of the solution; in one case Dr Murrell pushed the dose until 110 minims (more than a minim of pure nitro-glycerine) were taken at a time; but most patients are liable to experience alarming effects from fifteen to twenty minims. It gives rise to headache, a rushing noise in the ears, a sensation of fulness in the neck, and sometimes to nausea, languor, or drowsiness or even complete insensibility. These symptoms generally become more marked as the dose is increased; but with time a certain amount of tolerance of the remedy is often established. Delicate young women were found by Dr Murrell to be more susceptible to its action than most other patients.

The great use of nitro-glycerine, however, is in preventing the recurrence of seizures. In many cases it does this perfectly, the patient being able after a time to leave off taking it, and remaining apparently quite well and able to walk long distances and even uphill without discomfort. The plan which Dr Murrell advises is to give a dose of the remedy every three hours regularly, besides additional doses at each repetition of a paroxysm. I have, however, myself met with two cases in which persons thus freed from their symptoms ultimately died suddenly; and I think that anyone who has suffered from the disease in a well-marked form should always afterwards lead the most quiet life that he possibly can, avoiding all kinds of exertion and excitement. One of the two patients to whom I have just referred had returned to his business on the Stock Exchange.

Rest must, indeed, be regarded as an essential part of the treatment of angina pectoris. Probably all the general measures that I shall have to recommend in the management of cases of thoracic aneurysm find their application here also. Before the introduction of nitro-glycerine, arsenic was the medicine which proved most efficacious in warding off the seizures. In a good many cases it yielded very satisfactory results.

As an instance of the successful administration of nitro-glycerine in what Dr Walshe would doubtless have regarded as "pseudo-angina," I may relate the following case, which occurred to me in 1882. A bank clerk, about twenty-one years of age, had for some weeks been suffering from what he described as a "sudden tightness of the chest," which would stop him in walking, so that he could not walk more than thirty or forty yards. He experienced a pain at the lower end and a little to the right of the sternum as well as behind near the spine at about the same level. He also noticed that he could not lie on the right side in bed without discomfort. His own impression was that the seat of the affection was in the lungs rather than in the heart. I found that he had a pulse of 120, but this was in part due to nervousness, for it soon afterwards fell to 104. No sign of any organic cardiac affection could be detected. I prescribed for him one minim of the solution of nitro-glycerine three times a day. In about a week he lost his complaint entirely, so that (as I afterwards learnt) he took only a single bottle of the medicine containing twelve doses.

Fainting.—Syncope.—In the first chapter of this work I had occasion to describe the symptoms which accompany death by syncope or by failure of the heart's action; but it is necessary for me to return to the subject in this place, if only for the reason that in some cases in which the cardiac functions are for a short time completely arrested, there is no danger of a fatal issue.

I need not recapitulate in detail the symptoms of fainting which have already been given at p. 14, and which, indeed, are sufficiently well known. But it must be pointed out that they vary greatly in degree in different cases. Sometimes, after having suffered for some minutes from giddiness and nausea and "faintness," the patient just for an instant loses himself more or less completely and then gradually recovers. Sometimes he remains unconscious for a considerable time. Dr Walshe disbelieves in the possibility of recovery after the sounds of the heart have ceased to be discoverable during so long a period as five minutes. But it is of course not uncommon for "fainting fits" dependent upon an enfeebled action of the organ, to last for an hour or longer, and yet to end favourably. The subsidence of an attack is ushered in by "gasping or rather sighing respirations at long intervals, and by gradual return of pulse, consciousness, and colour. Sometimes vomiting or discharge of flatus, convulsions, or profuse perspiration takes place at the time of returning consciousness." Dr Walshe, from whom I have copied these details, thinks that the phenomena accompanying the re-establishment of life are generally painful and distressing, whereas he remarks that in very many cases the passage from life to death is rather attended by pleasurable sensations.

There are other instances in which the stoppage of the pulse and the interruption of the mental faculties are absolutely sudden, and in which the resumption of the heart's action and the recovery of consciousness are no less instantaneous, while there are no subjective sensations whatever. But although seizures of this kind are spoken of as "fainting fits" by unprofessional persons, they are, I believe, always really of cerebral origin, belonging in fact to the *petit mal* of epilepsy.

One great distinction between the attacks which are of an epileptic nature and those which may properly be referred to fainting, is that the former generally, if not always, occur without any definite exciting cause. On the other hand, the latter are commonly obviously traceable to some disturbing agency, though it may be apparently of a very trifling character; the heated air of a crowded room, the sight of blood (even from a cut finger), the strong odour of flowers, the introduction of a catheter, may each cause fainting in certain persons. It is especially apt to occur in young adults, in women rather than in men, and (above all) in those who are of nervous temperament.

Among the more frequent causes of the graver forms of syncope, such as are apt to prove directly fatal, are various organic diseases of the heart and aorta which will be fully discussed in subsequent chapters, pulmonary embolism, and the rapid withdrawal of ascitic or pleuritic fluid by tapping. The "cardiac" variety of sunstroke must also be mentioned. In this the sufferer, though he may probably himself be conscious of some premonitory symptoms, gives no sign of illness until he falls, gasps, and perhaps at once expires before anything can be done to help him. Dr Maclean says that this is the form most often seen in soldiers exerting themselves in the heat of the sun when dressed and accoutred (cf. vol. i, p. 770).

In the *treatment* of a fainting attack, the first thing is to place the patient in the open air or near a window and to make him lie down with the head as low as the shoulders; the clothes must be loosened about the throat and the chest, the crowding round him of sympathising friends must be prevented. A bottle of ammonia may be held to the nostrils, or if this be not at hand a bunch of feathers may be burnt so that he inhales the fumes. Cold water may be poured upon the face or the chest, the hands or the surface generally may be slapped with wet towels, a draught of cold water may be swallowed. If the stomach be overloaded an emetic of mustard should be administered; this, as Anstie says, "has a powerfully rousing influence upon the heart." Sal volatile, brandy, or ether may be given by the mouth, or either of the latter two may be thrown into the rectum in a small enema, or may be injected hypodermically.

Other measures, especially applicable when death seems to be impending, have been enumerated before (vol. i, p. 16).

AFFECTIONS OF THE MUSCULAR WALLS OF THE HEART

Dilatation and Hypertrophy.—*Anatomy—Diagnosis—Ætiology—Symptoms—Treatment.*
Fibroid Disease.—*Anatomy—Causes and pathology—Cardiac aneurysm—Symptoms—Course and event.*
Fatty Degeneration.—*Overgrowth and infiltration—Intrafibrous fatty degeneration: its histology, causes, and symptoms—Granular and calcareous degenerations—Rupture of the heart.*

The present chapter will be devoted to those organic affections of the heart which depend primarily on changes in its muscular substance.

Primary Enlargement of the Heart.—Hypertrophy and Dilatation.—We shall find hereafter that hypertrophy, generally attended with dilatation, of the heart is an almost inevitable result of all the more grave affections of the cardiac valves; and another very frequent cause of the same changes is Bright's disease of the kidneys. In each instance it is clear that the organ enlarges because the work which it has constantly to do is increased beyond the normal amount. There are, however, other cases in which cardiac hypertrophy is referable to an excess of bodily exertion. This, even if it is repeated day after day, has necessarily its intervening periods of rest; and the healthy heart naturally possesses a considerable amount of reserve force beyond that which is required to carry on the circulation in ordinary circumstances. Consequently, although the strain upon it is augmented by muscular efforts of all kinds, it is generally able to meet the call without suffering damage. Even if its walls should to some extent yield to the increased pressure which they have to bear they generally recover themselves afterwards, when the body returns to a state of rest, and the blood-current resumes its usual tranquil course. But such is not always the case. It sometimes happens that violent or frequently repeated bodily exertion leads to permanent enlargement of the heart. We must then suppose that one factor concerned in bringing about this result is a deficiency of power in the muscular fibres. Either they are perhaps naturally weak, or their strength may have been lowered by previous disease or by an insufficient supply of food. Again, it is probable that there are cases in which a like enfeeblement of its walls renders the organ unable to maintain even the natural tranquil circulation. I am not now alluding to the occurrence of fatty degeneration, or of other obvious organic changes in the fibres, for they will have to be described elsewhere. I rather refer to such a state of the heart as that which in the voluntary muscles is generally recognised as weakness, leading to fatigue on moderate or even slight exertion. In any case it seems likely that the immediate effect of the failure of the heart to perform its work efficiently is a yielding or *dilatation* of its walls, and that *hypertrophy*, or the increased growth of their substance, sets in afterwards. But, as a rule, it is not possible to trace this clinically.

The hypertrophy secondary to valvular lesions or to Bright's disease will be discussed in other chapters of thi swork. Here I shall confine myself to those cases in which enlargement of the heart takes place independently of such causes. But what I say with regard to the physical signs, and also with regard to the morbid anatomy and the histology of the affection, will of course apply equally to all forms of it, however produced. All the chambers often become enlarged together; but it is possible for some to be affected without the others, and separate descriptions must be given for the left and for the right side, at least so far as the ventricles are concerned. As regards the relation of hypertrophy to dilatation, writers are by no means clear in their statements. To me it seems that "simple hypertrophy" should mean an increase in the amount of the heart's muscle, its cavities remaining unaltered in capacity; "simple dilatation," an increase in their capacity, the amount of muscle remaining stationary. The former is, indeed, frequently observed, especially as a result of Bright's disease. And although the latter is perhaps never actually seen in the post-mortem room, it must yet be supposed to occur at the commencement of very many cases of heart affection. These definitions do indeed involve the necessity of admitting that a dilated heart is hypertrophied if its weight as a whole is above the normal standard, even though every part of its walls may be far below the natural thickness. No doubt it is in order to avoid this apparent inconsistency that Dr Walshe characterises as "simple dilatation" a condition in which "the walls are of such thickness as would be normal had the capacity been unchanged." But such a conception really rests upon an altogether arbitrary basis, because, for a dilated heart the normal thickness of walls should be proportionately augmented, in order to maintain its functional power; below this it is only a question of varying degrees of failure of "compensation." In fact although simple hypertrophy is in its physiological effects the very opposite of simple dilatation, yet in all the mixed forms of these affections it is to the latter rather than to the former that the case really approximates, however much increased the beats may be.

In the post-mortem room, then, in order to ascertain whether or not a heart is hypertrophied one has only to weigh it after having emptied it of its contents. According to Dr Peacock ('Reynolds' System,' vol. iv) the ordinary range of the weight of the organ is in males affected with acute non-cardiac diseases from nine to eleven ounces, with chronic non-cardiac diseases from eight to ten ounces; in females affected with acute disease from eight to ten ounces, with chronic disease from seven to nine ounces. But in large and powerful men who have been killed by accident or have died after a short illness, the weight may sometimes be as much as twelve ounces, or even more, without exceeding the limit of health.

It is much less easy after death to determine the presence of dilatation, at least in its slighter degrees; when it is well marked there can of course be no doubt about it. Dr Peacock gives the following figures as representing the normal dimensions of the two principal chambers:

	Males.			Females.		
	Lines.	Millimetres.	Inches.	Lines.	Millimetres.	Inches.
Circumference of heart . . .	103·7	233·32	9·209	104	234	9·236
Girth of right ventricle . . .	55·4	123·85	4·919	58·4	131·4	5·184
" left " . . .	48·3	108·67	4·289	45·6	102·6	4·049
Length of cavity of right ventricle .	43·3	96·42	3·821	44·3	99·67	3·925
" " left " .	37·6	84·6	3·333	37·1	83·47	3·197

But it is very difficult to make sure how far the apparent size of the organ is modified by the state of contraction or of relaxation of the muscular fibres. The difficulty arises in its extreme form in regard to what has been described as "concentric hypertrophy," a condition in which it is supposed that the wall of the ventricle grows in thickness at the expense of its cavity, this becoming actually smaller than under normal circumstances. But although some recent German writers are still disposed to admit that concentric hypertrophy occurs in some rare cases, I agree with those who doubt it. I have repeatedly in the post-mortem room seen hearts that at first looked as though they might be specimens of such a kind, but under a little stretching with the fingers they have always yielded and regained their natural size. Even when a hypertrophied heart seems to have moderately large chambers it still remains a question whether in a fully relaxed state of their walls they might not be larger; in other words, the pathological proof of the presence of hypertrophy without any dilatation is less simple than might be imagined.

It is only when the capacity of the cardiac chambers is not above the normal that one is justified in taking the thickness of their walls as proof of the presence or absence of hypertrophy. Dr Peacock gives the following as the normal measurements:

Thickness of the	Males.			Females.		
	Lines.	Millimetres.	Inches.	Lines.	Millimetres.	Inches.
Walls of right ventricle: base	1·85	4·16	·164	1·85	4·16	·164
„ „ „ midpoint	1·98	4·35	·176	2	4·5	·177
„ „ „ apex	1·42	3·19	·125	1·3	2·92	·118
„ left „ base	5·15	11·58	·425	4·9	11·02	·432
„ „ „ midpoint	6	13·15	·532	5·6	12·6	·497
„ „ „ apex	2·4	5·4	·214	2·5	5·62	·222
Septum between ventricles	5·73	12·89	·51	4·7	10·57	·421

But even when dilatation is present the determination of the thickness of the several chambers is of great importance, as enabling one to form some estimate of the degree to which the heart was competent to carry on its functions. In different cases there are very wide differences. One left ventricle measuring five or six inches in length may have its walls only two or three lines thick; another not more capacious may have them upwards of an inch thick.

As a rule, the weight of a heart dilated and hypertrophied independently of valvular lesions and of Bright's disease does not exceed fifteen or twenty ounces. But I have notes of one case in which it reached thirty-three ounces. Dr Peacock ('Reynolds' System,' vol. iv) speaks of hearts hypertrophied without any material valvular lesion or obvious source of obstruction in the aorta, and reaching the weight of from twenty-six to forty ounces. But I am not quite clear whether the presence of chronic renal disease was also excluded in these cases; in one of them, to which he refers as having been exhibited by Dr Bristowe at the Pathological Society in 1853, I should myself think it not unlikely that the state of the kidneys was really the cause of the cardiac affection, although this view was rejected after due consideration by Dr Bristowe himself.

In the cases which are now mainly under consideration we have no means of determining the rapidity with which the heart undergoes hypertrophy; probably the process is very slow and gradual. But when it arises secondarily to other lesions, of which the starting-point can be fixed, it has sometimes appeared to be much more rapid than could have been anticipated on *a priori* grounds. Dr Stone ('Lancet,' 1879) has related two examples of injury to the aortic valves by blows upon the chest, in each of which, if the heart was healthy at the time of the accident, it must have gained weight at the rate of nearly an ounce a week during the four or five months that elapsed before the patient's death. And Dr Goodhart ('Path. Trans.,' vol. xxx) has published a case, the history of which would suggest that the organ within three or four weeks grew to a weight of nineteen ounces, as the result of an attack of pericarditis. Dr Stone remarks that the increase in size of the pregnant uterus is less rapid than that observed by him in the case of the heart, namely two thirds of an ounce a week; but this assumes that the growth of the organ is uniform during the whole period of gestation, which is of course not likely to be the case.

There has been a good deal of discussion as to whether cardiac hypertrophy depends upon an overgrowth of existing fibres or upon a formation of new ones. Schrœtter (Ziemssen's 'Handbuch') cites Hoppor as having found the fibres to measure 0·03 mm. in a hypertrophied heart, as compared with a normal thickness of 0·007 mm. Friedreich also is said to have arrived at 0·025 mm. as the mean of ten measurements of the fibres of a hypertrophied left ventricle. But Rindfleisch says that he failed to discover any such difference, and his conclusion is that the fibres being unlike those of all other muscles in forming a network, undergo a further splitting up, which leaves them apparently of the same size as before.

I have already remarked that different cases seem to differ as to the order in which dilatation and hypertrophy are developed. In some instances it is probable that dilatation occurs first as the result of over-distension or of weakness of the cardiac muscle, and that hypertrophy follows later. In others hypertrophy appears to be the primary condition. In either case that which finally brings about a failure of the heart's functional activity is commonly the supervention of a further degree of dilatation, which causes the hypertrophy to be relatively inadequate. It is usually said that such an ultimate breakdown of the organ depends upon the occurrence of fatty changes in its muscular tissue. But Cohnheim ('Vorlesungen,' i, p. 72) throws doubt upon this view. There has, he says, been much exaggeration in the statements that have been made as to the frequency of fatty degeneration in hypertrophied hearts; and even when it is present he thinks it just as likely to be the effect of the disturbed compensation as its cause. His own view is that there is a simple "fatigue" or "exhaustion" of the fibres, unattended with any anatomical change that can be recognised with the microscope. Dr Allbutt ('St Geo. Hosp. Rep.,' vol. xviii) has drawn attention to a very interesting fact, which perhaps has a bearing on this question. It is that in the file cutters of Sheffield, who are constantly using the arm in rapid flexions, and in whom the *biceps* consequently undergoes great enlargement, this muscle after a few years again wastes, falling now far below its normal size. If the functional activity of hypertrophied muscles has thus a more or less definite period of duration it is obviously very important to relieve them, as far as may be, of all strains and extra calls upon them, so that the time when they are to break down may be postponed to the furthest possible limit.

The clinical *diagnosis* of hypertrophy and of dilatation of the heart rests partly upon percussion, partly upon inspection and palpation, partly upon auscultation.

By percussion one determines to what extent the heart comes into contact with the anterior wall of the chest uncovered by the lungs. Consequently, the results of percussion are modified not only by the state of the heart, but also by that of the lungs. If the lungs are emphysematous, the area of dulness due to the heart may be diminished, notwithstanding that the organ is enlarged; if the lower anterior part of the chest on the left side is flattened, or if the corresponding part of the lung is collapsed, the dull area may be increased though the heart is no larger than natural.

There is even a difference in the extent of cardiac dulness, according as the breath is drawn in deeply or forced out, so that for accurate percussion it is necessary that the breath should be held. A very good way of arriving at a satisfactory result is to make a series of marks with a soft pencil upon the patient's chest indicating the different points at which dulness begins to pass into resonance round the circumference of the organ. One thus obtains a more or less triangular figure, representing the region within which an absolutely dull note is obtained. Above it forms an angle, which in normal circumstances is situated at the upper border of the fourth left costal cartilage close to the sternum. From this point two lines are traced downwards, diverging in their course. One of them corresponds with the left border of the sternum; for although the right ventricle uncovered by lung lies behind the lower part of this line as far as the median line, the tone yielded by the osteal tissue when percussed masks the dulness that should theoretically be present and prevents any accurate definition of the space occupied by the heart on this side. Guttmann, however, says that the bone may be rendered less vibratile if the hand be laid over the upper part of it or if an assistant press firmly with the hands placed upon the rib cartilages on each side of it; in these circumstances a dull sound may be obtained. The other line extends downwards and outwards, passing to the inner side of the nipple until it reaches a point at which the apex of the heart can be felt beating. Along this line the transition from dulness to resonance takes place gradually, so that above it one can trace another line running more or less parallel and about half an inch distant, which indicates the upper limit of a region of partial dulness, just as the lower one indicates the upper limit of absolute dulness. The upper line begins above at about the level of the third rib. To complete the triangular figure, a base line has to be drawn from the lower end of the sternum to the point at which the heart's apex beats. It can scarcely be traced by percussion because the cardiac dulness passes insensibly into that caused by the left lobe of the liver. But Dr Gee says that sometimes a distinct heightening of pitch and increase of resistance can be made out in passing from one organ to the other. It must be added that, when the stomach is distended with gas, the resulting tympanitic sound is not infrequently transmitted by lateral conduction beyond the region which the stomach actually occupies. In fact, such a sound may often be elicited by tolerably firm percussion over the very spot beneath which the apex-beat can be felt. But in percussing over the heart it is essential that the stroke should be light and free and made from the wrist.

The statements that I have been making with regard to the natural extent of the cardiac dulness are applicable only when the patient is standing upright or lying upon his back. When he lies over to the left, the left side of the triangular area shifts further to the left, even though the heart may be of the natural size.

Now, when the heart is enlarged, the upper angle of the area of cardiac dulness commonly remains at the fourth rib. Dr Walshe, indeed, speaks of it as sometimes reaching even as high as the second rib; but I think that such a change in an upward direction is very unusual unless there is some morbid condition beyond hypertrophy and dilatation of the heart itself. On the other hand, the position of the two sides of the triangle varies widely from the normal. The right one may extend downwards along the right border of the sternum or even half an inch or an inch further still to the right; this is an indication of increase in size of the right ventricle. The left line may sweep outwards as far as the nipple or even still further to the left, and it is carried much lower than usual, the apex-beat being situated at a lower level in the sixth interspace or even in the seventh; this is a sign that the left ventricle is enlarged. The two ventricles are generally affected together; but if the right one alone is dilated, one effect of it is that the apex-beat is displaced outwards without being lowered. A pulsation may also be felt in the epigastrium. Dr Walshe states that distension of the right auricle contributes largely towards the increase of dulness to the right of the sternum at the upper part of the cardiac region. He says, too, that distension of the left auricle may cause abnormal deficiency of resonance in the third and second left intercostal spaces. And he even speaks of having recognised an impulse of presystolic rhythm in these several positions as the result of enlargement of one or the other of the auricular chambers. A necessary consequence of the displacement of the two lines marking the sides of the dull area when the heart is enlarged is that this area acquires a more or less definitely quadrilateral instead of a triangular form.

It is to be observed that for practical purposes nearly all that can be learnt from percussion of the heart, so far as concerns the diagnosis of enlargement of the left ventricle, may be gained by tracing one diagonal line from the fourth costal cartilage near the sternum to the site of the apex-beat, and another line at right angles to it from the lower end of the sternum upwards and outwards to the point at which the absolute cardiac dulness ceases. In normal circumstances the first diagonal line should not exceed two and a half inches in length; when the heart is much enlarged it may extend to four or five inches. The second diagonal line should measure about one and a half inches; it may be increased to more than two inches.

Now, it must not be supposed that percussion affords an infallible indication of enlargement of the heart, even when the lungs are healthy, being neither emphysematous nor retracted. On the contrary, very great hypertrophy of the left ventricle may be present (as for example in cases of Bright's disease) without there being any increase in the area of dulness. The organ seems to bury itself within the hollow of the left lung, so as not to come more widely into contact with the chest wall than in normal circumstances. One never has a better opportunity of demonstrating this fact than in cases of cerebral hæmorrhage; in such cases one often finds at the autopsy an enormously hypertrophied heart, notwithstanding that a few hours previously it may have been impossible to detect any clinical evidence of such a condition.

If percussion fails to reveal the presence of enlargement of the heart, I doubt whether any other results can be obtained from inspection, or from palpation, although it is certainly possible that the character of the apex-beat may be altered, notwithstanding that it is in its natural position. But great caution is required in diagnosing hypertrophy from this sign alone, at any rate while the action of the organ is disturbed by excitement.

When it has been determined by percussion that the heart is enlarged, the next question is whether the enlargement is due to dilatation or to hypertrophy. The solution of this question, so far as it can be solved, is to be found in a careful examination of the cardiac region by the eye and by the finger. In either case the heart's impulse may be seen and felt over a much more extended area than that of the natural "apex-beat." When there is great hypertrophy, it is often laboured and heaving, so that even a stethoscope with the observer's head resting upon it, is lifted as though by an irresistible power. In some cases, while the ribs immediately below the left nipple are pushed forwards, other parts of the chest wall are sucked in. On the other hand, when dilatation preponderates, the impulse may consist (to use the words of Dr Walshe) "either of a short feeble slap followed by a sudden fall back of the organ, or of a more prolonged faint tremulous motion." To the eye it has an undulatory character. Another peculiarity of the heart's action which indicates dilatation rather than hypertrophy is irregularity in the force of successive beats, or in their rhythm. Dr Walshe also remarks that there may be a want of perfect uniformity in the point at which the organ strikes the parietes. There has been some difference of opinion as to whether enlargement of the heart ever by itself causes an increased prominence or bulging of the præcordial region and a widening of the intercostal spaces, as compared with the state of the corresponding parts on the opposite side of the chest. Most observers admit that this is sometimes the case, especially in young subjects; but Schrœtter, following Skoda, maintains that it occurs only when there is also pericarditis, by which the textures forming the chest wall are softened. The latter opinion seems to me not at all likely to be correct.

Auscultation, I think, yields little direct information as to the existence of hypertrophy and of dilatation; its value lies rather in affording some guidance as to the state of nutrition of the cardiac muscle. To this I shall have to refer further on, when speaking of degenerative, fatty and other changes. But it is to be particularly noted that in cases of simple hypertrophy, with little or no dilatation, the first sound is often dull and muffled; Dr Walshe describes it as "prolonged and weakened, sometimes almost to actual extinction, the sensation reaching the observer's ear being rather one of impulse than of sound." When dilatation and hypertrophy occur together the first sound may be loud, higher-pitched than natural, and widely audible over the surface of the chest. But if with dilatation there is a soft, flabby state of the muscular tissue, the first sound may be very weak, faint, toneless, and of high pitch. Alterations in the second sound are not infrequently to be observed, but they depend upon the state of the arterial tension, and afford only indirect indications of the condition of the heart itself. The question whether a systolic murmur is ever due to dilatation of the ventricles, apart from the occurrence of regurgitation through the mitral or the tricuspid orifice, is a very difficult one, to which I shall have to revert hereafter, when discussing the significance of such murmurs. We shall find, however, that there is nothing to prevent our admitting that the cuspid valves may be rendered secondarily incompetent, either from failure of the papillary muscles to adjust their flaps properly, or as a mere result of the widening of orifices which necessarily takes place as the heart increases in size. Sometimes, however, the valves themselves appear to grow, so that they continue to bear a proper relation to the other parts of the organ. One character of a murmur due to failure of adjustment, and not to actual lesion of the valve, is said to be its inconstancy; it may be plainly audible one day and entirely absent on the next. But I rather doubt whether this is of much diagnostic value.

Sietz ('Deutsch. Arch.,' xi, xii) has drawn special attention to the fact that the movements of an enlarged heart within the pericardial sac are sometimes attended with sounds having a grazing or scraping quality, so as to be very like those produced by pericarditis. In one case observed by him, these continued up to the time of death, and no doubt was entertained as to the existence of pericardial inflammation; yet at the autopsy the serous membrane was found perfectly healthy, without any thickening or opacity of its surface. Dr Walshe, too, speaks of "knocking and rubbing additions to the first sound at the apex" as not very uncommon. I can myself recall one such instance when a diagnosis of pericarditis was made that on post-mortem examination proved to be incorrect.

Ætiology.—We have now to consider what are the *causes* of dilatation and hypertrophy of the heart, and in the first place I must repeat what has already been stated at the beginning of this chapter, that two of the most important causes are Bright's disease and affections of the cardiac valves. For in a great many cases it is impossible at the bedside to determine whether the state of the kidneys is such as to account for the occurrence of such enlargement, or even whether there is a primary valvular lesion—generally affecting the mitral rather than any other valve—to which it may be due. And even in the post-mortem room the very same points often remain doubtful, or at any rate the facts are so far ambiguous that different pathologists interpret them differently. Formerly, in cases of heart disease with dropsy, the diagnosis of "mitral regurgitation" was deemed sufficiently accurate, and physicians frankly admitted that they were often unable to tell whether the left auriculo-ventricular orifice would after death be found narrowed or widely open and "dilated." But now everyone recognises that in those instances in which it is dilated there is not in reality any actual lesion of the valve itself, but that the primary morbid process is an enlargement of the left ventricle. Still it may be held—and I think that until recently this was the common opinion—that regurgitation through the widened opening occurs secondarily, and constitutes the essential feature of the disease, being directly concerned in bringing about the lung induration, the nutmeg change in the liver, the dropsy, and all the other obvious symptoms. Of late, however, the tendency has been to disregard to a very great extent the presence or absence of mitral incompetency, and to look upon the impairment of the propulsive power of the ventricle as the really important thing in the cases in question. That is the view which I myself have long taught, and I even go further and entertain doubts as to whether a secondary mitral regurgitation really occurs so constantly as is supposed by those who regard a systolic apex-murmur as a sufficient proof of it. This, however, is anticipating another question which must be taken up further on.

As regards the kidneys, I shall have to show, when Bright's disease is under discussion, that these organs may be of good size and fairly healthy looking, and yet by the microscope may be shown to have undergone such extensive changes as fully to account for any enlargement of the heart that may be present. The renal origin of such cases is by no means always, or even generally, indicated during life by albuminuria, a low density of urine, or the presence of casts, even when the autopsy leaves no doubt about the matter. And there are a great many other cases in which, after the most complete histological examination of the kidneys, different pathologists would express divergent opinions as to whether the cardiac affection should be looked upon as secondary to the renal, or the renal to the cardiac. Another possible cause of enlargement of the heart has been recently found by some observers in extensive pleural adhesions. But I must confess to feeling uncertain whether the cases supposed to be of this kind which have recently been recorded by Bäumler and by Brüdi ('Deutsch. Arch.,' xix) really warrant the conclusions drawn from them.

Leaving now these intricate and doubtful questions, we have :—

(1) A clear and unquestioned cause of primary enlargement of the heart in *over-exertion* of the organ. Within the last few years its importance has been made manifest in various directions. I have already, at p. 8, cited Da Costa's observations on 'Irritable Heart' in soldiers of the United States army ; he states that in twenty-eight out of a hundred cases there was evidence of hypertrophy. In one of them death occurred from strangulated hernia eleven months after the commencement of cardiac symptoms; the left ventricle, though not apparently larger than natural, had its walls seven eighths of an inch thick at the thickest part. Fräntzel ('Virchow's Archiv,' 1873) has drawn attention to a like affection as having developed itself in nineteen soldiers engaged in the Franco-German war, especially among those who took part in the arduous march to Orleans, or in

the attack upon Belfort. He also refers to some statements, according to which recruits in the German army frequently become affected with hypertrophy of the heart as the result of prolonged and heavy marches, especially in summer. Fräntzel is disposed to doubt the correctness of these observations, but our own Army Medical Department has been for many years familiar with the prevalence of the disease among British soldiers even in time of peace. In 1870 Dr Myers published an essay in which he showed that cardiac affections in general were considerably more common in soldiers than in sailors, and in the Foot Guards stationed chiefly in London than in the men of the Metropolitan Police. And in a large proportion of cases he says that there is neither valvular disease nor disease of the aorta, but an extreme excitability of the heart leading after a time to enlargement of the organ. The general opinion formerly was that the cause lay in the crossbelts, heavy accoutrements, and tight clothing which the men used to wear, and by the urgent advice of Dr Maclean and Dr Parkes the old form of knapsack was abolished, and a "valise equipment" was adopted in its stead. Dr Myers laid especial stress upon the effect of the tightness of the tunic-collar in constricting the neck. It would be very interesting to know whether the prevalence of cardiac dilatation and hypertrophy among soldiers has been diminished within the last few years, but according to Prof. Veale ('Army Med. Dep.,' Report xxii) the necessary statistical data are wanting. He states, however, that this affection and the "palpitation" which is its most conspicuous symptom are still very frequent, and after careful inquiry into all the circumstances of a hundred cases, he assigns it in different instances to no fewer than seventeen more or less distinct causes. But it seems to me far more likely that some one cause is really responsible, and I am very much disposed to think that the real solution of the difficulty has been found by Surgeon F. A. Davy, who ('Army Med. Dep.,' Report xviii) refers it mainly to the "setting-up drill," during which recruits are compelled to "swell the chest" so as artificially to expand it. To this they are subjected for four hours a day during a period of about six months, having to march, and even to "double," with the chest in an abnormal condition. Dr Davy shows that in consequence of free expiration being prevented the functions of the lungs and of the heart must be very seriously interfered with, and he appears to have ascertained by direct observation that soldiers under the drill, even when they are standing, have the frequency of the respirations increased to about 40, and the pulse to 110 in the minute, that the heart's rhythm is often disturbed, and that the impulse of the organ is altered in position and more forcible, being felt over a wider area than natural. It is surprising to me that Dr Veale does not allude to this view of the matter, which appears worthy of the most serious consideration of the military authorities. Dr Myers had spoken of having often seen recruits perfectly exhausted after their morning's drill, which (one would suppose) ought not to have any such effect upon healthy young men. The notion is, of course, that the physique of the soldier is improved, the capacity of the chest being improved, but as Dr Davy remarks, this is of no advantage when obtained at the expense of its mobility.

Among civilians the ill-effects of over-exertion of the heart, though they had been cursorily alluded to by many previous writers, seem to have been first fully recognised by Dr Peacock in reporting, in 1864, upon the health of the miners of Cornwall, who, besides heavy hammerwork in the day, have to climb ladders of immense height in order to get out of the pit every evening. He found that many of them suffered from cardiac dilatation. Then came an important paper in the 'St George's Hospital Reports' vol. v, p. 23, by Dr Allbutt; but the cases that he observed hardly fall into the category with which we are now directly concerned, inasmuch as he assigned a very conspicuous place in the sequence of events to chronic changes in the aorta and in the aortic valves leading at length to aortic regurgitation. More strictly in point is a series of articles by Seitz in the 'Deutsch. Archiv' for 1873 and 1874. He showed that at Zürich cases are tolerably frequent in which during life there is great uncertainty as to the exact diagnosis of the cardiac affection from which the patients undoubtedly suffer, and which after death are best explained on the view that the disease is a primary enlargement of the organ. The patients have almost all been males, and engaged in heavy labour of one kind or another. But the most striking paper of all is perhaps one by Münzinger, in the 'Deutsch. Arch.' for 1877, on what he terms "the Tübingen Heart." It appears that at Tübingen heart disease, without any valvular lesion, is very commonly seen both in men and in women who work as labourers in the neighbouring vineyards, situated often upon the slopes of hills, up which heavy burdens of manure have to be carried. A point on which great stress is laid is that these poor people are very badly fed, living on potatoes and puddings, and scarcely ever tasting meat. Dr Allbutt too, alludes to insufficiency of food as an important factor in the ætiology of cardiac affections due to overwork and strain; he cites two cases of Dr Paget's which appeared due to the habit of taking long and active exercise while fasting; and he expresses the opinion that one reason why young men of the upper and middle classes do not more often suffer ill-effects from athletic sports is that they habitually live well. Thus it would seem that one must take into account not only the absolute amount of exertion which a person may have undergone, but also the condition of the cardiac muscle at the time. Muscular work of all kinds of course calls upon the organ for increased efforts to maintain the needful circulation. In strong, vigorous subjects there is a reserve force which is equal to all but the most excessive demands upon it. But in weakly, ill-nourished persons, the heart may fail under comparatively slight efforts. And there probably may be great differences in the vital endowments of the cardiac muscle itself in different individuals, altogether apart from the state of nutrition of the body generally.

It is obvious that these remarks are applicable likewise to hypertrophy and dilatation occurring as the result of valvular lesions or of Bright's disease. In such cases the amount of enlargement of the heart required to effect compensation for a certain degree of leakage, or to overcome a certain degree of obstruction to the arterial current, must largly depend upon the previous state of the cardiac muscles.

It is not to be supposed, however, that any change in the muscular fibres recognisable by the microscope is an essential feature of cases of enlarged heart from overwork. Some few fibres may be found fatty or granular, but such an appearance is altogether exceptional.

(2) Among patients belonging to the middle classes Traube ('Berl. klin. Woch.,' 1872, p. 223) was inclined to refer cardiac hypertrophy to "excessive smoking and to congestion of the portal system, resulting from sedentary habits and excess of food." The probably injurious effect of tobacco has been taken into account by all the writers who have discussed the ætiology of heart affections in the British army, but there seems to be no clear proof of its taking a prominent place among their causes. As for indulgence in eating, it doubtless falls under the category of those conditions which produce hypertrophy by increasing the arterial tension. These I must leave to be discussed elsewhere, when the relations between it and Bright's disease are under consideration.

(3) Since the recognition of the fact that anæmia induces a fatty change in the muscular fibres of the heart, it has naturally occurred to pathologists that those forms of the affection from which recovery takes place very pos-

sibly form the starting-points of subsequent cardiac disease. The question has been fully discussed by Dr Goodhart ('Lancet' i, 1880); but although he has shown that in women who are actually suffering from chlorosis the heart's impulse is diffused and displaced outwards, and that when anæmia proves fatal this organ is found to be dilated, he has not brought forward any clinical proofs of the development of permanent mischief from this cause.

(4) Very much the same may be said of the supposed production of enlargement of the heart by the exanthemata and other febrile diseases. Pyrexia is known to damage the muscular tissues, and Dr Goodhart has recorded ('Guy's Hosp. Rep.' xxiv) four or five instances in which sudden or nearly sudden death has occurred during scarlatinal dropsy and in which the heart has been found dilated or fatty. But in the cases in question it is difficult to say how much was due to increased arterial tension resulting from nephritis, and how much to the antecedent pyrexial state. And even if it be true that during enteric and other fevers the heart may become for the time dilated, it has yet to be shown that the organ is liable to remain in a morbid condition after convalescence. Prof. Veale, in his paper above referred to, on the causes of palpitation and cardiac disease in soldiers, says that the most common of them all is fever, chiefly malarial. But when he declares in support of this view that the physician must have had "either small experience, or very limited powers of observation who cannot call to mind many instances of permanent weakening of the heart after fever," he appeals to a court of which the verdict, I think, would at the present time be given against him.

(5) Another occasional cause of primary enlargement of the heart appears to be acute rheumatism. I have at any rate now and then met with cases which seemed to admit of no other interpretation. The most striking of these was that of a girl aged eleven, who died in Guy's Hospital of cardiac dropsy six months after a rheumatic attack. The most conspicuous lesion was dilatation of the left ventricle, which had reached such an extent that although its walls measured only from one eighth to a quarter of an inch in thickness, the organ weighed ten ounces. There had probably been regurgitation through the mitral orifice, for the papillary muscles were much wasted, but the valve itself was healthy or only slightly thickened. How such an effect is produced by acute rheumatism, apart from pericarditis, I do not know. Dilatation of heart may, however, be caused by extension of pericardial inflammation to the subjacent muscle.

Symptoms.—The symptoms of primary enlargement of the heart vary widely in different cases. In the earliest stage of the affection they consist partly in palpitation and subjective sensations of pain or discomfort in the cardiac region, partly in an increased frequency of pulse, which is often irregular or intermittent in rhythm. These phenomena have all been fully discussed in the last chapter. But another symptom, of which the absence is conspicuous in the merely functional diseases of the heart, is dyspnœa. This at first comes on during exertion only; the patient finds that he cannot walk so quickly as before with comfort, or that going uphill or ascending two or three flights of stairs makes him feel short of breath. From this condition there are all gradations up to a point at which even the slightest bodily movement becomes almost impossible. Inasmuch as in health muscular exertion makes the beats of the heart more frequent and calls for greater vigour of systole, there is no difficulty in understanding how it disturbs the action of the organ when diseased. And in cases of enlargement of the heart it has been specially noticed (as, for example, by Dr Veale among the soldiers whose cases he studied) that after even slight exertion or excitement the rate of the pulse becomes altogether disproportionately accelerated. But what is not so obvious is why this disturbance should give rise in the patient to a sensation of dyspnœa, even where the left ventricle is the seat of the affection, the right being healthy so far as can be ascertained. The explanation seems to be that in spite of its augmented frequency the heart after all fails to forward the blood through its left chambers with the needful rapidity; there is therefore an accumulation in the pulmonary vessels, and the right ventricle has to make increased efforts to propel its contents onwards. And as the result is a state of the pulmonary circulation identical with that which is produced in other cases by a deficient supply of air to the lungs, it is not surprising that the same feeling of shortness of breath is experienced. But the consequent increase in the frequency and depth of the respiratory movements would appear to be, in cases of disease limited to the left ventricle, rather prejudicial than useful to the patient, since it must still further augment the accumulation of blood in that chamber.

In many cases an early effect of cardiac dyspnœa is that the patient is unable to sleep with the head low. Instead of one pillow he has to use two or even three. In extreme cases he cannot lie down at all and is obliged to sit up in bed or to lean forwards. This condition is termed *orthopnœa.* In seems clear that the necessity for its adoption lies in the fact that it facilitates the descent of the diaphragm, which in the recumbent posture is hampered in its movements by the pressure of the abdominal viscera and especially of the liver.

In assuming, as I have done in the previous paragraph, that in cases of primary enlargement of the heart the left ventricle is the chamber earliest affected, I am not altogether in accord with recent writers on the subject, many of whom are inclined to think that the right ventricle often undergoes dilatation while the left still remains in a normal state. Thus Dr Allbutt relates in the 'St George's Hospital Reports' how on one occasion, towards the end of a long day's mountaineering in Switzerland, he was rather suddenly seized with a strange and peculiar *besoin de respirer* accompanied by a very distressing sense of distension and pulsation in the epigastrium; placing his hand over the heart, he felt a labouring diffused beat all over the epigastrium, and by percussion he ascertained that the right ventricle was very greatly dilated. He threw himself on the grass with his shoulders raised, and in a few minutes had the satisfaction of finding the distension, the oppression, and the dulness recede. He could then run and even move about on the level, but the instant he began to ascend the symptoms returned, so that it was only with great caution that he could proceed. During the following night he was awakened again with severe palpitation and dyspnœa, which, however, passed off as soon as he went to the window and drew a few deep inspirations.

In 1866 my friend, the late Dr Daldy, published a little work on 'Disease of the Right Side of the Heart,' in which, besides relating some similar cases to that of Dr Allbutt, he attributed to this cause a number of other symptoms that seem to be very doubtfully referable to it. I see no theoretical objection to the view that the whole of the heart takes part in the excessive strain produced by violent bodily exertion, and that if the right heart happens to be the weaker of the two, it may suffer before the left. But my difficulty is that, granting the occurrence of such cases, one ought, if they do not invariably end in recovery, to find them continuing up to the time of death as uncomplicated examples of enlargement of the right chambers, the left remaining of normal size and thickness. We are familiar with the fact that dilatation of the left ventricle leads to a secondary dilatation of the right ventricle; but it does not seem to be possible that the order of events can be reversed. All pathologists, however, are agreed that if those cases in which enlargement of the right side of the heart caused by pulmonary emphysema or by severe bronchial affection be excluded, such an affection is scarcely ever seen in the post-mortem room unaccompanied by a like affection of the left side. I myself remember to have met with but one case of the kind. It is that of a man, aged forty-one, who was admitted into Guy's Hospital under my care in 1880 on account of dropsy of the abdomen and legs. On examination I discovered a loud systolic murmur at the ensiform cartilage, musical in quality at that spot, propagated towards the right nipple as much as towards the left. On account of the rarity of primary disease of the right chambers, I hesitated to give this diagnosis, but the autopsy left no doubt about the matter. The heart, which weighed 16½ oz., was extremely broad and rounded in shape; the right ventricle was large and massive and had large fleshy columns, but the left one was quite small and flaccid; the right auricle formed the greater part of the base of the organ; the tricuspid orifice admitted more than five fingers; the edge of the valve was thick and opaque. Now, it had been a striking feature of this case that there was no orthopnœa; the patient, though very dropsical, lay quite low in his bed. I was disposed at the time to regard the easy state of the breathing as a further argument against the view that a cardiac affection of whatever kind was the cause of the ascites and of the anasarca; and it led me to think that the principal disease was perhaps of the liver, the murmur heard with the stethoscope being due to some accidental cause and having no real bearing upon the case. And I think that it has hitherto been the universal opinion that dyspnœa must necessarily be produced by any lesion of the right side of the heart interfering with the blood-supply to the pulmonary capillaries. But, on reflection, I am not at all sure that this supposition is well founded, and I look upon the point as one well worthy of consideration in future cases, and as possibly destined to aid greatly in the recognition of primary enlargement of the right ventricle.

Systematic writers upon diseases of the heart have been accustomed to draw a contrast, which does not appear to me to be well founded, between the effects of *dilatation* of the *left* side of the heart and those of *hypertrophy* of the same chamber. Dr Walshe, for instance, speaks of the pulse, in cases of pure hypertrophy, as full, tense, and resisting; he describes persons so affected as having a florid countenance and bright full eyes, and as liable to sensations of rushing of blood to the head, and to dull aching or throbbing cephalalgia. But these statements, however true of cases in which the cardiac affection is dependent upon an antecedent state of augmented arterial tension, as in Bright's disease, appear to me to be altogether inapplicable to those in which the causes of hypertrophy of the left side of the heart are such as have been described in the present chapter. The observations of Da Costa, Myers, and Veale, all point to the conclusion that whether hypertrophy or dilatation be the result of overstrain of the heart, the effect on the circulation (if any is noticeable) is always that it is retarded, or at least that its activity is impaired. The organ, in fact, becomes enlarged because in its natural state it is unequal to carry on its function; and the hypertrophy never passes beyond what is required for this purpose.

Prognosis and treatment.—The slighter degrees of enlargement of the heart subside when the cause of them is removed. Dr Allbutt's case is a good example to show how rapidly and completely an acute condition of dilatation may pass off, for it does not appear that he cut short his Swiss tour in consequence of the alarming attack that he had experienced. But when hypertrophy has developed itself, it is only as the result of long and patient treatment that a cure can be looked for. One most essential thing is rest. Among drugs the most useful appears to be aconite. Da Costa testifies most decidedly to its value in cases in which the cardiac impulse is unduly forcible; he generally gave one or two minims of a tincture three times as strong as the British tincture three times a day; or gr. $\frac{1}{60}$th—$\frac{1}{30}$th of aconitia. Dr Walshe speaks very highly of the same remedy; the dose which he recommends is gr. $\frac{1}{8}$th of the alcoholic extract of aconite. The usefulness of this medicine in cases of primary hypertrophy is not inconsistent with what has been stated above as to the symptoms of that affection, for when under the influence of rest and of good nourishing food the cardiac muscle recovers its tone and the dilated chambers resume their natural size, there is no longer any necessity for so forcible an action of the organ. In some cases veratrum viride may be employed with advantage; Da Costa administered drop doses of the fluid extract, or five to ten minims of the tincture, three times daily. Bromide of potassium is another remedy which is mentioned favourably by Dr Walshe. It does not seem that iodide of potassium is of any service.

If, however, rest be not taken in cases of overstrain of the heart, and if the early symptoms be neglected, the result is that sooner or later the hypertrophy fails in its purpose. Or perhaps there is from the first a condition of dilatation rather than of hypertrophy. In such circumstances digitalis is often of the greatest possible service. As its ultimate issue, the disease tends to pass into a condition of "imperfect compensation" or of "asystole," leading to a series of changes in the lungs and in the liver, and to dropsy of the dependent parts of the body, all of which will have to be fully discussed further on, as effects of valvular disease.

FIBROID DISEASE OF THE HEART.—Unlike fatty disease of the heart, which we have seen to be commonly more or less widely diffused throughout the substance of the organ, another lesion of the cardiac muscle, for which the best name appears to be that of *fibroid disease*, is in well-marked cases constantly limited to a definite area of the wall of the organ. Dr Quain has indeed described in his 'Lumleian Lectures' for 1872 ('Lancet,' 1872, vol. i) an affection which he terms "connective-tissue hypertrophy," in which he says that the muscular fibres are surrounded by connective tissue in all stages of its development—round-cells, spindle-cells, and bundles of fibrillæ. The thickness of the heart's wall is in such cases increased, but the most striking peculiarity is the firm, tough, leathery feel of its substance. Dr Quain says that slight degrees of this change have been overlooked, and he refers especially to a greatly enlarged heart, weighing forty and a half ounces, which had been for thirty years preserved as a specimen of cardiac hypertrophy in the museum of St George's Hospital, but in which upon examination the increased size was found to depend only in part upon an overgrowth of the muscle, the connective tissue being also greatly in excess.

With such an affection as this I must confess that I have no practical acquaintance. But on the other hand I have met with an exceptionally large number of instances of the localised fibroid disease, which I am now about to describe. No fewer than eleven such cases came under observation in the post-mortem room of Guy's Hospital in one period of less than twelve months (1873—74), and altogether I have notes of twenty-six cases that have occurred there at different times. In its slighter degrees it consists in the presence of streaks and patches of a milky white colour in the substance of the muscular tissue. The wall of the heart is thereby rendered more hard and resisting to the knife, but it may also acquire a succulent and spongy appearance, and when incised its cut surface looks uneven and rough. Microscopically there is seen a perfectly developed connective tissue, forming wavy bands, running in the same direction as the muscular fibres, some of which, or the remains of them, are still embedded in it. Or there may be a dense fibrous plate, looking like a piece of tendon, and consisting of a glassy substance with regularly arranged fissures or spaces, indicating the planes of fibrillation. It creaks when cut, and sometimes it contains calcareous salts in such quantity as to make it crackle under pressure, like an egg-shell.

The seat of this affection is sometimes the apex of the heart, sometimes some part of the anterior or posterior wall of the left ventricle, sometimes the septum. It never (I believe) begins in the right ventricle, but it may sometimes invade it by extension from the left. The fibrous substance is in some cases surrounded on all sides by muscular tissue, touching neither the endocardium nor the pericardium. It is then very likely to be overlooked, unless all parts of the organ are completely sliced up. In other cases it reaches one or both surfaces to a greater or less extent. The endocardium then shows a marked local thickening and opacity, while the visceral pericardium becomes covered with lymph, or may be adherent to its parietal layer. Some observers have thought that fibroid disease of the heart is generally secondary to pericarditis or to endocarditis, being the result of the spread of inflammation from the other tissues to the muscular wall of the organ. But this view is clearly inapplicable to many cases; and so far as the endocardium is concerned, there is an insuperable objection to it in the fact that an inflammation of this structure, apart from such an affection of the valves, is itself altogether unknown. As regards the relation between fibroid disease and pericarditis, it is to be noted not only that adhesion is often present only just where the fibroid material reaches the outer surface of the heart, but that even when the whole serous sac is obliterated, the two layers are often found to be very firmly connected together at that spot, but elsewhere so loosely that it is easy to separate them. Moreover, if extension from a general pericarditis occurred, one would expect to find connective tissue dipping into the muscle at a number of different points, which (so far as I know) is never the case. And in one instance I have known pericarditis develop itself so as to be recognised by physical signs about two months before death, at a time when the fibroid affection must have been already of long standing.

When the part of the left ventricle that is affected by fibroid disease includes the base of either of the fleshy columns of the mitral valve, the affection often spreads into the column, which becomes shrunken. But I do not think that one should include under the same category the very common cases in which the summits of the mitral columns undergo conversion into fibrous tissue by extension of growth from the tendinous cords attached to them. We may regard this as a probable cause of leakage into the auricle.

In all cases of fibroid disease of the heart there is considerable destruction of muscular tissue at the seat of the lesion. And when the whole substance of the wall is involved from one surface to the other, there is often not a trace of the normal structure to be discovered. The thickness of the wall is then generally much diminished. I only remember one case in which the result was to produce an obvious increase of it; in that instance the septum was the part affected.

Pathology.—With regard to the pathology of this morbid change there is still some uncertainty; perhaps it is not the same in different cases.

1. One view is that it results from a primary process of chronic inflammation, a *myocarditis*, arising in the cardiac muscular tissue spontaneously, or from rheumatism, or perhaps in consequence of a blow or fall upon the chest. At first, it is said, there is an infiltration of leucocytes; these subsequently develop into connective tissue. The atrophy and disappearance of

the muscular fibres at the seat of the disease is regarded as a secondary effect of the compression which they undergo. I have, indeed, never been able to discover a small-celled infiltration, even at the margins of the fibroid patches, in the cases that I have had an opportunity of examining; but perhaps the reason may have been that the morbid process was no longer advancing when the patient died. I find that in four of my twenty-seven cases taken from the post-mortem records of Guy's Hospital there was a history of a former attack of acute rheumatism. Another probable cause of chronic myocarditis is alcoholic intemperance.

2. In some instances *syphilis* gives rise to an affection which in its later stage is probably not distinguishable by its characters from fibroid disease due to other causes. At an earlier period it would doubtless be constantly characterised by the presence of gummata, as in eight cases cited by Lancereaux. It is worthy of notice that the development of gummata in the heart is by no means limited to the left ventricle; in two of the cases in question the right ventricle is said to have been alone affected, and in one the right auricle. Among my twenty-seven cases of fibroid disease of the heart there are four in which, from the presence of specific lesions elsewhere, the existence of syphilis could be safely asserted. In only one of them were gummata detected; these consisted of a mass as large as a bean within the wall of the right ventricle, and of a number of small, hard yellow points embedded in a reddish gelatinous substance at the growing edge of the fibroid material in the left ventricle. In one instance, the morbid process was limited to the septum, this being changed through nearly its whole thickness into a tough fibrous material with puckering and depression of the adjacent part of the endocardium. The disappearance of characteristic gummata in the more advanced stage of the disease corresponds perfectly with what is observed in the liver, in the testis, and in other organs.

3. The formation of *thrombi* in the cardiac cavities may give rise to an inflammatory change, extending through the wall of the part of the heart to which they adhere. I have once seen a well-marked instance of this in the auricular appendix. It is surely not improbable that the ultimate result might be the formation of a patch of fibroid disease. I have sometimes thought that such an origin might explain the frequency of fibroid disease at the apex of the left ventricle, for this point is very apt to become the seat of thrombi during the course of enteric and other fevers.

4. In a series of eleven cases of fibroid disease of the heart which I brought before the Pathological Society in 1874 there was one in which the cardiac muscle presented in addition what I described as a peculiar form of degeneration, consisting in its conversion into a dry-looking, greenish-brown substance, of the texture of wash-leather. At one part this formed a thin flat layer, embedded in the substance of the heart, and appearing as a narrow sinuous line in a vertical section of it. Under the microscope it was found to be merely muscular tissue, which retained its striation, and showed remarkably well the branching and reuniting of the fibres. Its characters contrasted strongly with those of the muscular fibres which lay within the area that had undergone the fibroid change; they exhibited but slight striation or had undergone fatty change. In the case of another patient, aged sixty-two, who afterwards came under my care in the wards of Guy's Hospital, and with regard to whom the question of the probable existence of fibroid disease of the heart was repeatedly discussed at the bedside, I found on post-mortem examination that the posterior wall of the left ventricle was much thinned, and was to a large extent converted into a lustreless yellowish-green substance, almost exactly like that just described; no fibroid material, however, was present. Thinking over these two cases, I have long seen clearly that the peculiar change in the muscular tissue must be the primary

affection, and that the fibroid development must be secondary, and I find that Dr Ormerod, twenty years ago, suggested ('Brit. Med. Journ.,' 1863) that the latter was due to a "process of conservative substitution, designed to fortify the walls of an attenuated and weak heart." But it is only quite recently that the true relation of the one morbid process to the other has been demonstrated to be as follows.

Weigert seems to have been the first to point out ('Virchow's Archiv,' 79, 1880) that fibroid disease of the heart is often the result of a change analogous to the formation of *infarctus* in other viscera. This has since been confirmed by the observations of Hüter ('Virchow's Archiv,' 89, 1882). He found precisely the same dry greenish or yellowish-brown patches as those which I had noticed, and traced them to obstruction of branches of the coronary arteries, sometimes resulting from thrombosis, sometimes from embolism, as in one case of endocarditis affecting the mitral valve in a young subject. And he recorded no fewer than eighteen cases of fibroid disease of the heart in each of which the affection was associated with sclerotic changes in the coronary arteries, corresponding more or less closely in distribution with that of the fibroid patches. At the same time he does not think that the formation of actual infarctus is a necessary step in the process by which coronary arterio-sclerosis leads to fibroid disease. It is sufficient that there should be a molecular change in certain of the muscular fibres, from interruption of their blood supply. The fibroid patches themselves he regards as the result of an inflammatory process set up by the disintegrating tissue elements. The relation between fibroid disease of the heart and *arteritis deformans* of the systemic arteries generally is one to which I myself drew attention in the 'Pathological Transactions' for 1874, and I commented on the fact that the former lesion was present in a very remarkable case, accompanied with absence of pulse in the limbs, which had been recorded by Dr Wilks, and to which I shall again have occasion to refer under diseases of the blood-vessels (p. 85). In the second of the two cases in which I found the greenish-looking patches now known to be infarctus, I noticed that the coronary arteries were extremely diseased, some of the branches being completely obliterated; but I could not make out that the two morbid changes were definitely related to one another.

Dr Wickham Legg, in his Bradshaw Lecture (1883), has thrown doubt upon the correctness of Hüter's view, and has expressed the opinion that the lesions found in the coronary arteries in his cases were only such as might have been anticipated from the advanced age of the patients. The importance of the peculiar change in the muscular tissue antecedent to the fibroid development seems not to have struck him. I can, however, quite confirm what he says as to the frequent absence of all obvious disease of the cardiac muscle notwithstanding that the coronary arteries are much obstructed. He cites a case of Dr Greenfield's, in which their orifices were indicated only by small vertical thickenings on the inner surface of the aorta; microscopically all that could be found was a slight but widely distributed fatty degeneration in the centre of many of the muscular fibres of the heart. In the year 1880 I met with three instances, in each of which arteritis deformans of the aorta had led to great narrowing of the mouths of both arteries and to almost complete obliteration of at least one of them. The cardiac muscles in these cases are either apparently quite healthy or at most a little soft and pale.

I am quite unable to reconcile Hyrtl's and Cohnheim's statement that the coronary arteries and their branches possess no anastomoses with the results of the injections made by Dr Legg and by Dr Samuel West, who found that they could readily fill one artery from the other, the two communicating by branches over the surface and the apex of the heart.

It might have been expected that the loss of contractile power of so much of the cardiac wall, inevitably resulting from the transformation of the muscular substance of the ventricle in its whole thickness into fibrous tissue should very seriously impair the efficiency of the organ. But Cohnheim has found experimentally that in the rabbit a large part of either ventricle, or even the entire lower third of the heart may be held fast in a clamp so as to be completely deprived of its function without the arterial pressure becoming at all lower in consequence. And experience shows that fibroid disease sometimes gives rise to no symptoms whatever; in three of my twenty-seven cases in which it was discovered at the autopsy the patient had died from some other cause. On the other hand, the frequency with which dilatation and hypertrophy are associated with this morbid process proves that in many instances the systole of the ventricle is more or less interfered with; in ten of my twenty-seven cases the heart was considerably enlarged, weighing from twenty-one to thirty-five ounces. I think that a peculiar alteration in the shape of the ventricular cavity is produced by the presence of fibroid disease in its wall; it becomes deepened from before backwards so that the mitral valve lies much further from the anterior surface of the heart than usual. I have seen the valve separated from the posterior surface also by a considerable interval.*

Cardiac aneurysm.—During the systole, any portion of the wall of the heart that has undergone the fibroid change must be exposed to great pressure; one can easily imagine it forming a "protrusion" like those that are observed in the frog's heart when the action of digitalis is beginning to manifest itself, as was described by Dr Stevenson and myself in a paper read before the Royal Society in 1866. Indeed, when the whole thickness of the cardiac muscle is destroyed, a permanent yielding of the affected part almost always results. Sometimes it forms a shallow pouch, sometimes a sac of greater or less size, communicating with the ventricle by a comparatively narrow opening. Aneurysm of the heart, in the strict sense of the term, appears in fact to be seldom if ever anything else than a complication of fibroid disease of the organ. It is of course to be understood that I am not now referring to a mere dilatation of any one of the cardiac chambers (which in the early part of the century was often called aneurysm), nor to the destruction by ulceration of a part of the wall of the heart in connection with a like affection of the valves (which is still sometimes spoken of as acute aneurysm). Leaving these affections out of consideration, I may confidently say that in all but an insignificant minority of cases, cardiac aneurysm depends upon a pre-existent fibroid change in the muscle. Dr Legg in his Bradshaw Lecture ('Med. Times and Gaz.,' 1883, ii) has cited three cases which appear to show that it may be produced by fatty degeneration; but there is at any rate no doubt that such a result from that cause is quite exceptional. The reason doubtless is that in fatty hearts the morbid process is too widely diffused, and the ventricular systole too feeble, for any great pressure to be thrown upon any one part of the chamber. So, again, although aneurysm of the heart, like fibroid disease, is met with at all periods of adult life up to a very advanced age, I am under the impression that the latter change is much more often found without any yielding of the ventricular wall in old people than in those who are middle-aged or young. Among Hüter's eighteen cases of fibroid disease, only four of which occurred in patients under sixty years of age, there were but four in which aneurysms were present; the ages were fifty-six, sixty-two, seventy-three, and eighty. Both affections are very greatly more common in males than in females. Cardiac aneurysms do not usually attain a very great size, but sometimes they become as large as the fist. They then of course project from the surface of the heart; but the smaller ones, especially if there are several of them, are sometimes excavated within its substance. I have recorded ('Path. Trans.,' 1874) a remarkable instance in which the wall of the left ventricle was tunnelled out in all directions into cavities, of which the largest was as big as a walnut. Fibrin is often deposited in large quantities in the interior of the sac of an aneurysm of the heart; in the specimen just referred to many of the cavities were filled with an adherent greenish gelatinous substance, containing curd-like whitish degenerating flakes. Dr Wilks has placed on record ('Path. Trans.,' vol. viii) a case in which there was found attached to the apex of the heart a cured aneurysm of the size of a pigeon's egg which had calcareous walls, and the interior of which was completely consolidated. I well remember the autopsy, for it was the first at which I was present within the walls of Guy's Hospital when I entered as a student there in October, 1856.

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* [See on the histology of fibroid disease of the heart, a valuable paper by Dr F. C. Turner, in the 'Trans. Internat. Med. Congress,' 1881, p. 427.—Ed.]

Symptoms.—Clinically fibroid disease and aneurysm of the heart can very seldom, if ever, be diagnosed. The presence of the former affection should perhaps be suspected when cardiac symptoms without evidence of valvular lesions are present in a patient suffering from senile gangrene or showing other signs of general arterial disease. A venereal history, too, may lead one to infer that the heart is or has been the seat of gummata, out of which a fibrous tissue ultimately has developed, and it is very important not to overlook the possible existence of syphilis as a cause of disease of this organ, on account of the good results that may be anticipated from a prolonged course of mercury and of iodide of potassium. One striking instance of this has recently come under my notice, and Dr Balfour may be cited in testimony that cases of "excited action of the heart with hypertrophy" in his practice have yielded to antisyphilitic treatment. The physical signs in cases of fibroid disease of the heart are, in fact, undistinguishable from those of an enlargement of the organ due to overgrowth of the muscle. In either case, a systolic apex murmur may or may not be audible; at one time it may be present but not at another. When there is a very large sac projecting from the heart's apex, a careful mapping out of the area of cardiac dulness might possibly suggest the real nature of the case; but the disease would still have to be diagnosed from an aortic aneurysm pushing downwards into the region ordinarily occupied by the organ. In one instance Skoda is said to have observed bulging of an intercostal space overlying the seat of a cardiac aneurysm. The symptoms of fibroid disease of the heart are sometimes those of obstruction to the pulmonary and systemic circulation, including dropsy; they were so in nine among my twenty-seven cases. In one instance the period that elapsed from the beginning of the patient's illness up to the time of his death was remarkably short, being only seven weeks. But other cases have run a protracted course, and have at first been benefited by treatment with digitalis and diuretics, for that which impedes the blood flow is, after all not the fibroid affection itself, but the failure of compensation on the part of the wall of the ventricle in general. In a few instances it has been noted that the pulse has been unusually slow, from 28 to 48 in the minute.

In a great many cases, however, the heart has gone on discharging its functions quite naturally, so far as can be known, until the patient has suddenly fallen down dead. For example, Dr Whipham ('Path. Trans.,' xxi) has recorded the case of a gentleman, aged twenty-nine, who fell dead from his horse while riding in Hyde Park, having started in good spirits and apparently perfectly well, and having never before exhibited any symptoms of cardiac disease. The abrupt stoppage of the organ in such circumstances is at present altogether unintelligible. But it is perhaps worthy of notice that there is an exact parallel for it in the results of the experimental ligature of one coronary artery, or even of a large branch of one coronary artery, in the dog, as practised by Cohnheim ('Virch. Arch.,' vol. 85). After this operation the heart for a little while goes on beating with perfect regularity and maintains the arterial pressure at its normal level. But at the end of about ninety seconds its pulsations become somewhat less frequent and their rhythm is slightly disturbed, and about half a minute later both ventricles suddenly stop at the same instant, after which no stimulus whatever succeeds in restoring their contractions. Evidently, therefore, it is not sufficient, in searching for the cause of sudden death, to examine the orifices and main trunks of the coronary arteries. An arrest of the blood flow through any one of the principal branches must be supposed capable of accounting for it, and equally so whether there be embolism, or a more chronic change in the vessel, leading gradually to the same result.

FATTY DISEASE OF THE HEART.—(1) *Adipose overgrowth and infiltration.*—The natural layer of adipose tissue beneath the pericardium is often found increased in elderly persons, and within limits this may be regarded as a physiological condition which gives rise to no symptoms during life. It chiefly affects the right side of the heart, especially at the base of the ventricle.

But sometimes the fat grows in upon the muscular fibres so as to thin the cardiac wall, and this becomes a cause of atrophy of one or both ventricles. Occasionally the *adipose ingrowth* may penetrate right through the wall until it meets the endocardium, and this is particularly seen near the apex of the right or the left ventricle.

Again, there may be *fatty infiltration,* the adipose tissue increasing between the fibres. A large amount of interstitial fat in the muscles of the limbs, as in cattle fatted for the market, and in men who drink largely of beer, is probably indicative of over-feeding and under-work. When it affects the diaphragm, it may become more serious. But when the cardiac tissue is so infiltrated, as frequently occurs in conjunction with adipose overgrowth and sometimes alone, it is probable that, though no certain signs or symptoms of its presence arise, it may be the immediate cause of death. Obesity is notoriously a bad condition for recovery from surgical operations and other injuries, and a "weak heart" is often the cause of the want of repair and fatal issue.

(2) *Fatty degeneration.*—This is a different pathological condition. The heart may be free from adipose tissue, but its tissue is pale, soft, and flabby. On the inner surface, particularly of the musculi papillares of the left ventricle, pale yellow zigzag markings are seen, described in the 'Med.-Chir. Trans.,' vol. xxxiii, by Dr Quain as "tabby degeneration." Under the microscope the fibres are found to have lost their striæ, and instead black granules appear, at first in transverse lines, as if the change has affected disk after disk. Next the dark granules become larger and acquire a bright glistening centre, and all trace of structure disappears.

According to Dr Hermann Weber,* the amount of etherial extractives from such hearts is not greater than normal. Dr Stevenson, however, as quoted by Wilks and Moxon, found the fatty matter nearly doubled, and Krylow has apparently settled that there is a decided relative increase, though much less than one would have anticipated.

The most frequent causes of this fatty degeneration of the heart (which is quite independent of obesity either of the heart or other parts) are (1) anæmia, (2) certain poisons. It is most constant and well-marked in those remarkable cases of idiopathic anæmia first described by Addison, and since known under the name of "anémie grave" and "perniciöse anämie." It is also often found in cases of leuchæmia and Hodgkin's disease, and occasionally in phthisis, cancer, and other wasting diseases. It is a constant appearance in fatal cases of phosphorous poisoning when the liver is also the seat of remarkable fatty degeneration; and the same thing has been observed in poisoning by arsenic, and (I believe) by mercury and lead. Lastly, acute fatty degeneration often occurs as a superficial change immediately beneath an inflamed pericardium.

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* "Zur Lehre von der fettigen Entartung des Herzens," 'Virchow's Archiv,' xii, 326 (1857). Dr Weber has favoured me with a statement that subsequent extension of his inquiries confirmed his previous results. The wasting of the normal adipose tissue and the small extent of the degenerative process, often limited to the left ventricle, may explain these results.

The *symptoms* of true fatty degeneration are very obscure and doubtful. It is usually surmised from our knowledge of pathology rather than diagnosed by physical signs.

The cardiac impulse is described as being weak but irritable, or sometimes "slapping," *i.e.* distinct but short. The first sound is often accentuated and has lost its booming character so as to resemble the second. The radial pulse may be quite unaffected. General symptoms of lividity, dyspnœa, irregular pulse, &c., probably only appear when there is concomitant dilatation.

When a person past fifty, pale and thin, with a white, soft, and "satiny" skin, and early arcus senilis suffers from dyspnœa, and his heart gives a short, sharp first sound, one may form a diagnosis of fatty degeneration; even without the presence of the graver forms of anæmia or of atheromatous or valvular disease of the heart and arteries.

The event of fatty degeneration is undoubtedly in not a few cases sudden and fatal syncope. Often, however, it is found post-mortem when death has occurred in other ways. It does not appear in itself to lead to dilatation, to which it is rather secondary; but it certainly may end in rupture of the heart.

A brown or *granular degeneration* of the cardiac muscles has been frequently observed, but its relations to fatty degeneration and its pathological significance are still obscure.

The rarest form of cardiac degeneration is that which has been described by Köster as *calcareous infiltration.* A good account of it, with two original specimens figured is given by Dr Coats in his recent 'Manual of Pathology' (p. 318).

Rupture of the Heart.—This rare and interesting pathological condition owes its practical interest to the importance of discriminating it from rupture as the result of injury. In the latter case the lesion is almost always in the right ventricle or one of the auricular appendages. In idiopathic rupture it is almost always in the left ventricle. In most cases the muscle is already weakened by fibroid, fatty or granular degeneration; but several instances are on record in which no such changes have been detected.

The rent does not always go through the entire thickness of the wall and may in such cases possibly be recovered from; but usually hæmorrhage into the pericardium ensues from the torn vessels, and causes death as certainly, though not so rapidly, as when a larger rent opens directly into the ventricular cavity.*

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* [The paragraphs upon this and the preceding page have been added by the Editor.]

PERICARDITIS

Rarity of idiopathic pericarditis—Its antecedents—Anatomy—Physical signs—Symptoms—Events—Adherent pericardium—Treatment of pericarditis.

Ætiology.—I shall have hereafter to speak of pericarditis as a complication of acute rheumatism, and, in truth, it is seldom idiopathic. Sometimes acute pericarditis has followed prolonged bodily exertion, for example, after a long march, or it has apparently been set up by exposure to cold, generally with pleuropneumonia. Scarcely any acute disease is more rapidly fatal than a double pleuropneumonia with pericarditis, but in these cases the pulmonary symptoms override those of the pericardial inflammation. Pericarditis is sometimes the first manifestation of acute rheumatism, pain and swelling of the joints coming on only when it has existed for two or three days. Hence, when a patient dies of pericarditis after a very short illness, one can hardly exclude the possibility of a rheumatic origin of the attack. But this explanation applies especially to children, in whom such a rheumatic pericarditis preceding any affection of the joints is much more common than in grown-up persons.*

If we exclude those cases in which inflammation of the pericardium is set up by wounds or direct mechanical injuries, we find that it can generally be traced to one of two causes—either (1) to some general morbid condition, or (2) to a pre-existing local disease of a neighbouring part.

(1) Next to acute rheumatism, Bright's disease of the kidney is the most common precursor of pericarditis. In Russia pericarditis has been often observed to occur in scurvy, but this has not been confirmed by English physicians. Gout has also been mentioned as a cause, but this perhaps has acted only indirectly through the renal disease which so often complicates it. Tubercular deposits may set up pericarditis, but this is not commonly observed at the bedside, and occurs chiefly when several serous membranes together are attacked by tubercle, independently of the organs covered by them. Formerly pyæmia was said frequently to give rise to this and to other serous inflammations, but it is now believed that pericarditis occurs in pyæmic cases only when suppuration has first attacked the heart's substance; the affection would thus come under our second head.

(2) Pleuropneumonia (of the left side especially) is often associated with pericarditis, but generally the disease attacks both organs simultaneously, and therefore cannot be strictly said to pass from one to the other. Mediastinal tumour or abscess, aneurysm of the root of the aorta, disease of the ribs or even of the mammary gland, are other causes of pericarditis, and it not uncommonly arises intercurrently in cases of cardiac dropsy towards the end of life.

Anatomy.—The morbid appearances characteristic of pericarditis are in part the same as in other serous inflammations. In an early stage the membrane becomes minutely injected and loses its lustre. Then lymph appears upon its surface, often first around the roots of the great vessels. As this lymph increases in amount, however, it assumes appearances which differ from those ordinarily seen in pleuritis or peritonitis. It forms thick concentric layers over the heart's surface, which may be stripped off in succession.

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* [See, for rare cases of Idiopathic Pericarditis, Prof. Bäumler's paper, in the 5th vol. of the Clinical Soc. 'Reports' (1871). I once saw an uncomplicated idiopathic case with Dr Dalton, of Norwood, in a healthy man about 40.—Ed.]

The parietal pericardium likewise becomes lined with lymph, and between the two surfaces there is more or less serous or sometimes sero-purulent liquid. In consequence of the incessant movements of the heart, the surfaces now become remarkably roughened. Sometimes they bristle with a number of papillæ, more often they look honeycombed, or resemble the interior of the paunch of a ruminant. Another good comparison is that made use of by Laennec, and afterwards by Hope, who say that the surface looks like that which would be produced by squeezing some butter between two flat pieces of wood and then suddenly separating them. So shaggy does the heart sometimes look in these cases that the name of *cor hirsutum* was formerly given to it. Rather more than a century ago Haller described the "hairy" heart as occurring especially in bold and adventurous men.

In other cases advancing pericarditis leads to the effusion of more and more fluid. When the disease is acute this apparently cannot much exceed the amount that can be injected into the sac after death, viz. twelve to eighteen ounces; when this limit is reached the diastole of the ventricles is interfered with, and great distress is produced, terminating in the rapid death of the patient. But in chronic cases a much larger quantity of fluid may accumulate in the pericardium, more than three pints having sometimes been found. Suppuration appears to be much less frequent than in other serous sacs, but this probably depends rather on the preponderance of rheumatism as a cause than on any special tendencies of the membrane itself to one morbid change rather than to another. In very rare cases the fluid in pericardial effusion has been known to undergo decomposition with the evolution of fœtid gas. This condition, *hydro-pneumopericardium*, somewhat less infrequently arises from the extension of disease from a mucous surface (as that of the œsophagus or stomach) to the serous sac.

The inflammation often extends from the pericardium to adjacent parts. Thus pleurisy may be set up, especially on the left side. In other cases the mediastinal tissues become affected, so that the parietal pericardium is fixed to the sternum by dense adhesions. The areolar tissue above the heart may participate in this change, and the left innominate vein may have its coats very greatly thickened and its cavity plugged with coagulum, a point of some clinical interest, as accounting for the occurrence of œdema of the left arm, altogether independently of any tendency to general dropsy.

Another very important extension of pericarditis is to the heart. The outermost strata of muscle are soft, and of a pale yellow or dull greyish-red colour.

Signs.—The diagnosis of pericarditis in practice turns upon the discovery of the physical signs of the disease, and can scarcely be based upon the other symptoms taken by themselves. The earliest of these signs is generally the friction-sound or rub. It is true that in some cases, even before this can be heard, the onset of pericarditis may be suspected from the heart's action becoming disturbed and tumbling, and the first sound noisy and prolonged, but even then the detection of a rub first converts this suspicion into a certainty. A pericardial rub sometimes lasts for weeks, the disease leading to little beyond the effusion of lymph; in other cases it rapidly disappears, being audible only for a few days, or even only for some hours. This often is due to the fact that the two serous surfaces have become separated by fluid, and consequently no longer rub together as the heart moves. The presence of this fluid is indicated by special signs, the most important of which is an increase in the area of the cardiac dulness, and in practice it is generally found that the augmented dulness is first discoverable at the base of the heart. Instead of the percussion-note in the third left interspace being but little less resonant than at the corresponding point of the opposite side, it may be completely dull, and this dulness often reaches as high as the second rib and has been known to extend above the clavicle. When the quantity of fluid is considerable the left lung is pushed to one side and compressed, and dulness on percussion may exist over so large a part of the left side as to cause the case to be mistaken for one of pleuritic effusion. Another sign of pericardial effusion, if extensive, is bulging of the præcordial region with widening of the intercostal spaces, and the diaphragm may even become depressed and the epigastrium prominent.

Even comparatively small quantities of fluid generally suffice to separate the heart from the chest wall, and its impulse may consequently be diminished or imperceptible, but this sign is very far from being constant. On the other hand, an impulse can often be felt in the fourth interspace, slightly external to the line of the natural "apex-beat."

In general it may be said that the positive value of the physical signs above enumerated is very great; friction-sound is conclusive as to the presence of lymph, and increased dulness upwards, if developed during an attack of acute illness, proves that liquid has been poured out into the pericardial sac. On the other hand, I am by no means sure that the absence of a pericardial rub can be regarded as disproving the existence of acute pericarditis when there is other severe disease. I have more than once failed to discover pericarditis a few hours before death in cases of double pleuro-pneumonia in which the heart has been found covered with recent lymph.

Symptoms.—These vary remarkably in different cases; and in Bright's disease pericarditis is often altogether latent. Pain in the cardiac region and in the epigastrium may be most intense and agonising, and it may radiate widely over the chest, and down the left arm to the elbow; pressure over the heart or in the pit of the stomach may cause the greatest distress. But in other instances the patient feels no pain, nor is there any tenderness. It was formerly maintained (in this country by Addison) that pericarditis is painful only when it is associated with pleuritis, the pericardium itself being insensitive both in health and disease. But I believe that no positive evidence has been brought forward in support of this view; and certainly pleuritis is often present when pain is by no means severe.

The heart's action, again, may be regular or irregular, quiet or greatly disturbed, and attended with palpitation very distressing to the patient. When the symptoms already referred to are present in an intense degree there is generally much dyspnœa; the patient can hardly speak for want of breath and because of the tightness of the chest; his features are anxious and drawn; his nostrils dilate with each inspiration; he generally reclines on his back, with his head raised; but it is remarkable that when copious effusion has occurred, he often lies by choice flat in his bed, with scarcely a pillow, the least elevation of the head producing a tendency to syncope.

Most writers on the subject mention the occasional occurrence of violent cerebral disturbances in acute pericarditis. Thus maniacal delirium, rapidly fatal, has sometimes been the principal symptom; and the case has been regarded as one of cerebral inflammation, until the autopsy showed that the pericardium was the seat of disease. But similar cerebral symptoms occur in acute rheumatism, independently of pericarditis, with very great elevation of the temperature; and this fact raises the question whether, when pericarditis is present, it is really concerned in the production of the delirium. The same may also be said of the choreic movements occasionally observed in pericarditis, for there is a very close relation between acute rheumatism and chorea. Again, apoplectiform stupor, hemiplegia and convulsive attacks may perhaps be traced to embolism of the cerebral vessels, consequent on attendant endocarditis.

Dysphagia has been mentioned as a symptom of pericarditis; and has been referred to the pressure on the œsophagus by the sac distended with fluid. Walshe disputes this opinion, and refers it to a nervous or dynamic origin.

Event.—It is a rare thing for acute pericarditis in any of its ordinary forms to be the sole or even the direct cause of death. By Louis the average mortality was estimated at one in six cases; but in the pericarditis of acute rheumatism the immediate danger is very far less than this. In Bright's disease, death often follows quickly upon the occurrence of pericarditis as a complication; but even then we may often doubt whether it has been much concerned in causing the fatal result. Sometimes, however, fluid effusion accumulates in so large a quantity as to hamper the heart, apparently interfering especially with its diastole.

When recovery is about to take place the symptoms gradually subside, disappearing generally in about twelve to twenty days from the commencement of the disease; the fluid effusion diminishes by absorption, and if the corresponding surfaces of the heart and parietal pericardium are still roughened by lymph a *redux rub* may be heard on auscultation. After a time this lymph also in great part disappears; but before this occurs the pericardial surfaces are commonly glued together to a greater or less extent, and become permanently adherent. There has been much difference of opinion as to whether such adhesions necessarily occur in every case of pericarditis in which lymph has been effused. Some of the best observers think so, but I should for my own part have been disposed to doubt it.

Adhesions.—The physical characters of pericardial adhesions vary greatly in different cases. Sometimes, especially after the lapse of a long time, they are reduced to a mere film of connective tissue, which the fingers can tear through with but little difficulty. In other cases they are exceedingly tough, so that the only way to denude the heart is to strip off all the tissues superficial to the muscular fibres. Then, again, they may be uniformly of great thickness; or they may include masses of altered lymph, accumulated in certain parts of the pericardial sac, and especially round the great vessels. Lastly, the inflammatory material may in course of time undergo calcification, and the heart thus appear to be enclosed in a bony case.

This condition of obliteration of the pericardial sac by adhesions did not escape the notice of the older pathologists, but it was then supposed to be a congenital defect. Since its real nature has been understood physicians have contended whether it is or is not of clinical importance. And the general conclusion appears now to be that this depends on the quality of the adhesions. Thin areolar connections appear not to hamper the heart's movements in any way, but a thick mass of hard fibrous tissue surrounding the organ may give rise to serious symptoms. By Hope and others it was formerly maintained that an adherent pericardium always tended to cause hypertrophy of the heart's substance; but it is now known that this was a mistake. If one or more of the chambers is hypertrophied in such a case, this is the effect of some previous disease or of a coexisting valvular affection. On the other hand, the presence of thick pericardial adhesions is often associated with atrophy of the ventricular walls. This may in part be the result of the myocarditis which often accompanies pericarditis rather than of the adhesions themselves, but it is precisely in such cases that an adherent pericardium most often gives rise to symptoms and that it can (if ever) be detected by physical signs.

The early auscultators attempted to diagnose adherent pericardium by various signs which were not long in being shown to be fallacious. At the present time the point on which most stress is laid is the occurrence of systolic depression at the site of the impulse, while one or two intercostal spaces above this recede at the same time. Slight retraction of the spaces close to the sternum during the systole is by no means uncommon even when the pericardium is healthy; but it appears probable that obliteration of its cavity may generally be inferred when a considerable region of the chest wall is drawn in. Still, however, we may doubt whether this can occur without the pleura over the heart being adherent and the left lung being withdrawn from its natural position, and if so, it might perhaps be met with as a result of old pleurisy apart from any pericardial adhesion.

Some observers have also endeavoured to diagnose pericardial adhesion from the fact of the heart's dulness not being diminished during inspiration or from the position of the organ remaining unaltered when the patient lies on different sides; but these signs are still more uncertain.

Treatment.—The treatment of pericarditis, according to modern practice, is far from being active, and would have been regarded as very inadequate by the earlier auscultators, who sought to recognise the disease at the earliest possible moment, chiefly in order that they might combat its progress by the so-called antiphlogistic measures. At the present day venæsection is never employed, and if leeches are used, it is with the object of relieving distress and dyspnœa, for which purpose they have undoubted value. Mercurials are scarcely used. The influence of mercury on inflammation is denied by many of the most competent observers, and pericarditis offers little opportunities for testing its value, since the natural duration of the disease varies greatly in different cases. The therapeutical measures which we now adopt are as follows:—The patient is kept very quiet in bed, the præcordial region is covered with a poultice, a thick layer of cotton wool, or a hot flannel; light fluid nourishment is given to him with a saline or effervescing mixture. Opium or chloral is prescribed for pain and restlessness. A blister is also very useful in relieving pain if the disease be not in a very acute stage, and I have seen the same application very efficacious in removing effusion; at any rate it has been followed by a rapid diminution of the dulness which for several days before had remained much more extensive than natural. Iodide of potassium is frequently given, with the hope of favouring absorption. I am not aware of much evidence of its value in this disease, but its undoubted efficacy in some allied affections is a sufficient ground for employing it.

When urgent dyspnœa and threatening suffocation arise from the presence of pericardial effusion the operation of paracentesis should be performed. This was suggested more than two centuries ago by Riolanus, but it appears to have been first practised by Romero, of Barcelona.* A slight incision is first made, and the trocar is then inserted gently into the fourth or fifth left intercostal space about an inch away from the sternum so as not to wound the internal mammary artery. Aran even injected iodine into the pericardial sac, after removing about two pints of fluid; and the patient recovered. The operation has now been performed in a sufficient number of cases to make it quite warrantable, and when, for instance, as sometimes happens, the pericardium is found after death enormously distended with pus, we regret that paracentesis has not been done. One of the most successful cases has been published by Dr Samuel West. The patient was a lad of sixteen, who had been suffering from increasing dyspnœa for three weeks until he applied for admission at the Victoria Park Hospital. The physical signs led to the belief that a large pericardial effusion existed, and he was accordingly tapped. Fourteen ounces of pus were withdrawn. In a few days the same amount was again taken away, but the fluid still re-forming, an incision was made into the sac through the fifth interspace and a drainage-tube inserted; two quarts of purulent fluid were removed. The patient gradually recovered and remained quite well ('Med.-Ch. Tr.,' vol. lxvi).

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* [Successfully. This was in 1819. Quoted, in a summary of fifty cases, in a dissertation on "Paracentesis Pericardii," by Hindenlang, 1879.—Ed.]

INFLAMMATORY AFFECTIONS OF THE HEART

Myocarditis—*Rheumatic and otherwise secondary—Pyæmic.*

Acute Endocarditis—*Benign form—Ulcerative or infective form—Physical signs.*

Chronic Endocarditis and Valvular Lesions—*Anatomy—Causes—Effects on the heart—On lungs, liver, and other organs—Valvular murmurs—Rhythm—Seat—Conduction—Differential characters and relative prognosis—Non-valvular murmurs—The pulse in valvular disease—Sphygmographic tracings—General symptoms—Treatment.*

Myocarditis.—Inflammation of the muscular tissue of the heart may take place under special conditions, but it is only positively recognised when it occurs in connection with endocarditis or pericarditis. In the latter case it is not at all unusual to see the layer of muscle immediately beneath the serous membrane involved in the inflammatory process; it is pale and soft, whilst the microscope shows it to have undergone a change in texture of a granular or fatty kind. In some exceptional cases the whole thickness of the walls may be seen to be affected, and in these a dilatation is apt to occur which may lead to speedy death or become a permanent morbid condition. At times long subsequent to the inflammatory attack its effect may be seen in what is called a fibroid disease of the heart, a condition in which the walls of the ventricle show on section numerous white fibrous streaks interspersed with the muscle. This has already been treated of above (p. 33).

A more acute inflammation of the muscle, or rather one of a different kind, is met with in pyæmia, in which numerous small abscesses may be found scattered through the tissue of the organ. A single circumscribed abscess is sometimes met with in connection with endocarditis and disease of the valves. The diagnosis of myocarditis can only be conjectural.

Endocarditis.—By this is generally understood an inflammation of the valves and the adjacent parts on the left side of the heart. The usual cause is rheumatism. The inflammation is recognised by the increased vascularity of the serous membrane and by an exudation which causes some roughness of its surface and which again leads to a deposition of fibrin on the valves. It is to these that so many of the consequences of endocarditis are due. It is this lodgment of fibrin on the inflamed surface which constitutes the peculiarity of endocarditis and causes it to differ from inflammation of other parts. The living blood is always ready to deposit fibrin on a roughened surface and especially when an excess of fibrin exists in it and the current is in any way checked. Moreover, as Dr Moxon has conclusively shown, these fibrinous concretions again set up inflammation and ulceration in neighbouring parts by their rubbing against the surface; indeed, most important changes in the heart are due to the friction of these vegetations by their aggravating and extending the inflammatory process.

The first indication of endocarditis is seen in redness and vascularity of the valve, afterwards by the production of a row of granulations along the lines where the valves meet. These are bead-like elevations formed along the delicate curved line on each side of the corpus Arantii where the sigmoid valves touch, and also along the corresponding edges of the mitral or (rarely) the tricuspid valve. If examined microscopically these nodules are found to consist of ordinary inflammatory cell-elements. The whole body of the valve may also become infiltrated with these inflammatory products, and so being swollen and softened is ready to undergo still further changes. In recent endocarditis these granulatious along the valves are the first and only indications of the inflammation. At a later stage they become larger and confluent, and hanging down into the stream of blood, attract coagulating fibrin, which then collects upon them in large quantities. It is thus seen that vegetations on the valves have a double origin, being in part derived from the valve itself and having therefore a true inflammatory source, and in part derived from a deposition from the blood; the two products running into one another and not being clearly separable. When these vegetations are long and move about they come into contact with the surface of the ventricle and there set up a fresh inflammatory process; so that the part touched is also soon covered with a patch of vegetation. In this manner a vegetation on one valve may bore a hole through another with which it comes in contact. There are few cases of acute inflammation of the heart in which these secondary effects are not seen, and of the valves, the mitral seems more likely to be affected than the aortic.

Effects.—Subsequently the texture of the valves becomes involved, the tissue is loosened, and the valves become much altered in structure. The inflammation may go on to *ulceration*, and a rent or perforation may take place so that a complete hole may form in the aortic or mitral valve, which then becomes surrounded by vegetations deposited from the fibrin of the blood. At other times an aortic valve, may be found partially detached from its walls, or the chordæ tendineæ of the mitral ulcerated and broken; their loose ends floating about and covered with fibrin. As a consequence also of these inflammatory changes, thickening and adhesion or coalesence of the valves may take place leading to obstruction at the orifices, or on the other hand retroversion of the valves leading to reflux or regurgitation.

Acute *ulcerative endocarditis* is not so common an affection as the more chronic process, and is often due, as Dr Moxon has shown, to the effects of friction of fibrinous concretions on the surface. These rub against the wall of the cavity and there produce an ulceration followed by a further deposition of fibrin, or, proceeding further onward, invade the muscle until an abscess is formed. This finally discharges its contents into the heart and constitutes an *acute aneurysm*. It usually occurs at the root of the valve, and it may sometimes reach the surface of the heart, when if it bursts it sets up a fatal pericarditis. Although the fibrinous concretions spoken of are probably the usual instruments in the production of the aneurysm the latter may arise independently of them and as a result of the more simple inflammation. In some cases of ulcerative endocarditis the inflammatory products become detached, and infecting the blood, set up a fatal blood-poisoning; it is probable that in these cases the concretions undergo a change, and it is said also that organisms are frequently met with in them, as first shown by Heiberg.

The result is infectious embolism causing "pyæmic" abscesses of the spleen, kidneys, and other viscera, including the heart itself, and aneurysms of distant arteries.*

Signs of endocarditis.—The presence of endocarditis is indicated by the occurrence of a bruit heard over the region of the heart in connection with rheumatism and other acute disorders. If, for example, in acute rheumatism a bruit arise near the apex of the heart we suppose that inflammation has

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* [This arterial pyæmia as it was entitled by Dr Wilks, has lately been ably treated by Dr Osler, in his Croonian Lectures (1885), as Malignant Endocarditis.—Ed.]

been set up in the neighbourhood of the mitral valve. If it occur at the base we believe it may be associated with inflammation of the aortic valves; in the latter case the diagnosis is less certain, as bruits of a temporary character are often met with in acute rheumatism which are probably hæmic. The fact of the disappearance, however, of the bruit by no means warrants the positive denial of endocarditis, since there is good reason to believe that an inflammation of the valves may be recovered from; witness the case of the disappearance, on recovery from chorea, of the bruit which in many cases appears to be due to an organic change in the valve. Sibson made the observation (and it has been corroborated by others) that, preceding the occurrence of a bruit in rheumatic fever, the first sound may often be observed to be prolonged. Occasionally a diastolic bruit suddenly occurs in the course of rheumatic fever. This implies that the inflammation of the aortic valves has caused ulceration and laceration of one of the segments.

Chronic Changes of the Valves.*—These changes may arise out of the acute or be essentially chronic in their nature. One of the commonest changes is for the valves to become thickened by a growth of connective tissue which by subsequent contraction considerably alters their shape; and calcareous matter also is frequently deposited in them. On these chronically diseased valves vegetations may arise, so that it is often difficult on examining a specimen to decide whether the malformations are the result of a process which was from the first acute or whether the more marked acute process has not supervened on a previously thickened valve.

In the case of the *mitral valve* the effect of the chronic disease is very often to produce stenosis or narrowing of the aperture; the wall of the valve, especially towards its free edge, is thickened and the segments may closely cohere. Sometimes there are deposited in it masses of calcareous matter, giving rise to the old name of "ossification." The chordæ tendineæ may undergo a similar change and coalesce so that each papillary muscle gives origin to a single large fibrous column, which may be more or less fluted, or pierced with one or two slits indicating the line of separation between the chordæ of which it was made up. At the same time the cords become much shortened, so that the edge of the valve is drawn down and assumes a funnel-shaped form, or more rarely appears as a kind of diaphragm between the auricle and ventricle with a small hole in its midst. This is usually styled the "button-hole mitral," and more particularly so if the opening is in the form of a slit. This may be so narrow as scarcely to allow the insertion of the tip of the finger, instead of three fingers, which ought readily to pass through a healthy valve. It is a remarkable fact in cases of stenosis of the mitral valve where much thickening is observable and where a history of rheumatism and endocarditis might have been expected, that often no such evidence can be discovered: the symptoms sometimes can be traced back to childhood and in some cases the disease may have been congenital. It is possible, however, that thickening of the valves, being of a very slow growth, might have arisen during some of the slighter forms of rheumatism when endocarditis was not suspected. Stenosis of the mitral is much more common in women than in men and is a form of disease always to be suspected in childhood if the heart is affected.

Inflammation of the mitral valve need not, however, always produce stenosis, but the reverse; the chordæ tendineæ may become elongated, and so, being unable to preserve the valve in a due state of tension, allow it to become retroverted towards the auricle; or without retroversion by the mere lengthening of the cords the due apposition of the segments is prevented and the valves rendered inefficient. It must, however, be remembered that regurgitation through the mitral valve, owing to its inefficient closure, by no means implies structural changes in the valve, since it often arises from over-distension of the ventricle.

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* [From this point to the end of the present chapter, the author's unfinished MS. has been continued by the kindness of his senior colleague Dr Wilks.—Ed.]

In the case of the *sigmoid valves of the aorta* similar changes may take place; the edges may unite or all three valves join together to form a funnel with an aperture at its end. The attached borders between the valves become obliterated so that only a line of projection is left to indicate the original segments. Many good authorities have regarded these cases of stenosis from adhesion as congenital. In other cases a valve may become thickened and distorted so that it hangs down in the aorta a mere shapeless mass. This of course produces regurgitation. Sometimes the valve is retroverted; at other times it is perforated, or may be found dragged down as if forcibly torn away from the aorta. It may here be remarked that in cases of regurgitation both of the mitral and aortic valves there may often be observed a reticulated patch of fibroid tissue on those parts of the auricle or ventricle on which the regurgitant stream of blood has impinged.

The *valves of the right side* of the heart may be affected in the same way, though more rarely. The tricuspid may occasionally show a thickening of its curtains and sometimes be narrowed into a buttonhole. The pulmonary valves are very rarely affected.

Ætiology.—As regards the causes of the changes in the valves an endocarditis accompanying rheumatism is the most common; a similar inflammation may, however, be observed sometimes in pyæmia and scarlatina, and the result of an endocarditis is not infrequently seen in chorea, manifested by a fine row of beads along the meeting edges of the mitral and aortic valves, more especially the former. There can be no doubt that the chronic changes found in the aortic valves are frequently brought about by hard labour, especially when the exertion has been of that kind where the arms are rhythmically thrown backward and forward as in rowing or sawing; this is the reason why aortic disease is so rare in women compared with men, and that when it is observed in the former a history of acute rheumatism is nearly always found. It was noticed by older writers that aortic disease was met with much more frequently among sawyers than other labourers, in consequence of their work being done in a pit by lifting a heavy saw.

Although it is universally admitted that hard labour and exercise with the arms of a peculiar kind will in course of time cause damage to the valves it has never been generally accepted that violent exertion may cause a sudden injury to a valve, from which all subsequent organic alterations may spring. The question is constantly arising in consequence of patients who are the subjects of valvular disease maintaining that they were perfectly well until on a given occasion they made some unusual exertion by attempting the ascent of a hill, running to catch a train, or, as it was said by two patients, mounting a lofty cathedral spire; after these unwonted feats they became breathless, were unable to advance further, and ever since have been the subject of distress which the medical man had recognised as cardiac. The difficulty in arriving at a just conclusion arises from the fact that these persons are usually middle-aged and had never tested the integrity of their organs before the occurrence of the dyspnœa, and also from the fact that if death occurs two or three years afterwards and the heart is carefully examined, any supposed injuries or lacerations are so changed by subsequent inflammatory processes that it is impossible to declare what is new and what old.

Effects of valvular disease on the heart.—It can be easily understood how the deranged mechanism of the heart due to disease of the valves must react on the muscular walls of the cavities and necessarily disturb their normal working power; it is also manifest that this derangement must affect the circulation throughout all the organs of the body. It is highly important to be cognisant of these changes during the life of the patient since they are productive of marked and peculiar symptoms. It may be laid down as a rule, that obstruction in any cavity or passage causes hypertrophy in the muscular structure behind it, as seen in the case of the bladder in stricture or in the intestine in a chronic obstruction; in like manner stenosis of the aortic orifice produces hypertrophy of the left ventricle, and stenosis of the mitral a like hypertrophy of the left auricle. Regurgitation through a valve causes enlargement of the cavity, seeing that it is being continually over-distended; it follows therefore that the left ventricle is dilated in aortic regurgitation and the left auricle in mitral regurgitation; in the latter case the enlargement is not excessive, seeing that the blood is thrown back on the lungs, which have to bear in part the pressure. It is evident that any obstruction on the left side of the heart must affect the lungs and so react again on the right side of the heart. For example, in cases of primary pulmonary obstruction, as in chronic bronchitis, where the blood flows with difficulty through the lungs, the right ventricle of the heart becomes hypertrophied and as a natural consequence the right auricle also; there is thus produced the well-known hypertrophy of the right side of the heart in bronchitis, the texture of which may be noticed to be remarkably tough. If again we take the case of mitral stenosis, we find that the left auricle, in order to propel the blood through the narrow orifice, has more work thrown upon it and becomes in consequence much enlarged and hypertrophied and at the same time its lining membrane opaque and thickened; the blood in the lungs in like manner must be retarded and the right side also becomes enlarged as in bronchitis. Sometimes in stenosis, owing to the whole current of blood being reduced to the measure of the mitral orifice, the left ventricle becomes somewhat diminished in size, and the whole heart smaller than in health. In the cases of aortic disease, in which there is an impediment to the flow of blood as well as regurgitation, the left ventricle becomes hypertrophied and dilated to a very great size. This impediment to the escape of blood from the aorta arrests the flow behind, and therefore the left auricle participates in the enlargement and as a consequence the right side of the heart also. In such a case the whole heart is immensely enlarged and is often called "bovine." It frequently weighs forty-five ounces, and even much heavier hearts than this are sometimes met with.

Effects upon other organs.—It may be convenient to mention in this place the changes which take place in other organs from the long-continued congestion. In the case of the *lung* this may be so great that blood is brought up during life, and the organ is found after death to have blood effused into the tissue; this may be scattered through the substance or seen as large round circumscribed dark masses, constituting what is called "pulmonary apoplexy." These usually occur at the lower parts of the right lung. If the blood do not actually burst through the vessels, the engorgement goes on until the capillaries are completely blocked and the alveoli of the lungs become almost obliterated; in consequence of this the lung becomes very dense, hard, and fleshy, and sinks in water, the cut surface being smooth. This condition is sometimes called "splenization." Sometimes, after a time, exudation takes place, a large quantity of pigment is formed, the alveoli are thickened, and the lung becomes dark and granular; this is styled "brown induration." If more marked inflammatory products are thrown out, and some formation of connective tissue takes place, the consolidation which then arises, combined with the granular pigment, produces an appearance to which the term "brown indurated pneumonia" has been given. A close examination discovers the remarkable changes which have occurred in the vessels of the lung due to the long-continued pressure; sometimes they have become varicose, and as regards the pulmonary arteries atheromatous degeneration of their coats is frequently seen.

The *liver*, which from the long-continued engorgement becomes much enlarged, is found to be in the state denominated "nutmeg." The arrested flow of blood in the hepatic veins produces a similar congestion of the portal circulation as well as a stagnation of bile in the ducts, the capillaries become choked, fatty degeneration of the cells takes place, and the secretion becomes arrested. The appearance of the liver is altered in a most characteristic manner, the fatty degeneration of the circumference of the lobule giving it a white border, and this white being mixed with the red of the blocked hepatic veins and the yellow of the obstructed ducts produces the nutmeg condition. It is still a question whether production of new connective tissue may also take place. Several authors speak of this condition going on to cirrhosis, so that in course of time a structural disease of the liver is produced. This, however, has never been thoroughly proved.

The *spleen*, in like manner, is found in cardiac disease to be hard.

The *kidneys* are large, hyperæmic, tough, and coarse-grained.

The *stomach* is intensely injected, and its surface covered with mucus.

When portions of fibrin have been washed from the heart, wedge-shaped masses of so-called infarction are found in the spleen, lungs, and kidneys.

Altered sounds arising from disease of the valves.—These are called "murmurs" or "bruits;" they are of various kinds, one of the most common being the bellows murmur or *bruit de souffle;* others are harsh, sawing, or grating, and some musical. As to the cause of these morbid sounds there has been until lately a generally accepted opinion that they were due to friction, that is, that the blood pouring over a roughened surface or passing through a narrow channel set up a new sound by the rubbing of its particles against the irregular edges. This was not, however, the original explanation, and of late years the older doctrine has come into vogue as more reasonable and more in accordance with clinical facts. One of the original investigators was Corrigan, who says, "When an artery is pressed upon the motion of the blood in the artery immediately beyond the constricted part is no longer as before. A small stream is now rushing from a narrow orifice into a wider tube and continuing its way through the surrounding fluid. The rushing of the fluid is combined with a trembling of the artery and the sensation to the sense of hearing is the *bruit de souffle.*" This doctrine, long put aside, has been more recently supported by Chauveau, who by means of various experiments has shown that when blood passes into a dilated vessel a sonorous jet is produced and that this vibration of the current causes the bruit. The sound, therefore, is produced, not at the point of obstruction, but just beyond it, and this is what Corrigan had already pointed out in cases of stenosis of the aorta. The same explanation applies when a bruit is produced by the pressure of the stethoscope on an artery causing an artificial constriction. And to the same cause is due the *bruit de diable* heard in the jugulars of anæmic subjects where the blood passes into a large dilated vein. In the case of constriction of the mitral orifice the conditions are most favourable for its production, the blood passing through a narrow aperture and then breaking up into a number of jets in the larger ventricle beyond. Various illustrations have been given from other physical phenomena in corroboration of the doctrine: for example, when water runs swiftly through a narrow orifice it is not at this spot that a sound is produced; but it is beyond, where the stream breaks up into a number of smaller ones that the noise is loudest. The bruits therefore are not now regarded as due to friction between the blood and the walls, but as due to oscillations in the blood itself or friction amongst its particles as it passes through channels of unequal calibre. The sound would of course be influenced by the rapidity and force of the current, and this is what is observed in the case of the heart, the sounds varying in intensity in proportion to the strength of the propulsion.

The commonest bruit is that of which we have been speaking; others have been described under the names of *bruit de scie* and *bruit de râpe.* The vibrations which cause these bruits may sometimes be felt by the hand like the purring of a cat, and this thrill was denominated by Laennec *frémissement cataire.* Besides these bruits we meet sometimes a musical murmur (*bruit d'oboe*) there being a sufficient number of vibrations produced to constitute a musical note. The theory for the production of the souffle does not seem here applicable, and therefore it is supposed that some string or edge vibrates in the blood as the stream passes over it. The musical bruits are usually attendant on marked valvular disease and may sometimes be heard some distance from the body, and very readily down the arms or legs, which act as conductors or natural stethoscopes. By placing the fingers of the patient in the auscultator's ears, and thus converting him into a double stethoscope, such a musical sound is often well heard.

The presence of a bruit denotes the existence generally of disease or imperfection of a valve, but the nature of the affection is discovered by other means, viz. (1) by the rhythm of the bruit or murmur, (2) by its position or seat and by the direction which it takes.

Rhythm.—In considering the action of the heart, it must be remembered that the physiologist has divided its movements into closing and opening, or into systole and diastole, and that during the former the first sound is heard, and during the latter the second, due to the closure of the sigmoid valves. The first sound is therefore styled *systolic* and the second *diastolic,* but it must be distinctly remembered that the closure of the aortic valves is only one of the events occurring during diastole, this taking place at its commencement, and that the filling of the heart and the contraction of the auricle also occur during diastole. The heart's action may be looked upon in this wise. The ventricle is a large hollow muscle placed in the course of the circulatory vessels to propel the blood continually onwards; after its contraction the aorta takes on the movement and so in this way keeps up the onward flow of the blood. Behind the ventricle is placed the auricle, which may be regarded as the dilated termination of the vein acting as a receptacle to supply the heart. The action, therefore, of the whole series is that of contraction of auricle, ventricle, and aorta in regular series. The blood is seen to flow along the veins into the auricle and through the open valve into the ventricle, then the auricle suddenly contracts, followed by the ventricle, and afterwards the aorta contracts. The contraction of the auricle is noiseless, but that of the other parts, ventricle and aorta, produce the first and second sound. It must be distinctly noted that the series of contractions is complete before it begins again, that is, the aorta has ceased to contract before the auricle comes into action, and therefore the contraction or systole of these two parts is not synchronous. It was a want of knowledge of this, or a direct belief to the contrary by Laennec and others, which so long led to a misinterpretation of the different bruits. Inasmuch as both auricle and aorta contract during the diastole of the ventricle, any sounds occurring during these times are called diastolic, but nevertheless they are not synchronous, the aorta contracting at the beginning of diastole and the auricle at the end. To avoid, therefore, confusion in terms we are in the habit of saying that any sound produced during the contraction of the auricle or that part of the diastole which is not occupied with the contraction of the aorta is *præsystolic.*

If the time of the complete action of the heart be divided into fifths, about two fifths are occupied by the contraction of the ventricle, one fifth by the contraction of the aorta, and nearly the remaining two fifths by the diastole or filling of the auricle and ventricle. The short period of time not occupied by this is taken up by the quick contraction of the auricle.

If we watch the movements of the heart in a living animal we observe a deliberate movement of the ventricle followed by that of the aorta, but the auricle so momentarily contracts before the ventricle that it looks as if the movement of the one passed into that of the other by a kind of screw-like motion. The whole action of the heart indeed is screw-like, the ventricle is spiral, and as it contracts the blood is sent spirally into the aorta, which then takes on the same action, and the sigmoid valves close down, not simultaneously, but one after the other in a similar spiral manner. This spiral movement of the ventricle is due to the arrangement of six or seven distinct layers of muscular fibres crossing one another. The use of the papillary muscles is clearly for keeping the curtains tight during the contraction of the ventricle.

If we now take the three cavities in succession, auricle, ventricle, and aorta, and consider that the valves between these separate cavities may be variously injured, and that new and morbid sounds would thereby be produced, we can easily discern at what period of time these bruits would take place. We have already said that these valves may be affected in such a way as to cause either obstruction or allow regurgitation, and as we have mainly the left side of the heart to deal with, we have merely to consider the mitral valve between auricle and ventricle causing obstruction or allowing of a reflex of blood, and then considering the aortic valves in like manner. In the first place, it is obvious that if any obstruction at the mitral orifice caused a bruit, the latter would be auricular in rhythm, or, as it is usually called, præsystolic. If, in the second place, the mitral valve was so diseased as to allow reflux, the sound so produced would occur during contraction of the ventricle and therefore would be systolic. Thirdly, if disease of the aortic valve were productive of obstruction and a bruit occurred, the latter would take place during the contraction of the ventricle and be systolic. Fourthly, if the disease of these valves allowed regurgitation of blood through them and a bruit were produced during the contraction of the aorta, it would for the reason before named be styled diastolic. In considering, therefore, the more ordinary diseases of the valves, the bruits produced by them would be four in number, one auricular, two ventricular, and one aortic, or, in clinical language, one presystolic, two systolic, and one diastolic. All these may be developed on both sides of the heart, making eight in number, but they are mostly produced on the left side only, the presystolic being due to obstruction of the mitral valve, the systolic murmur either to obstruction of aortic valve or regurgitation through the mitral, and the diastolic to regurgitation through the aorta.

The seat of the bruits or murmurs.—The valves of the heart are placed so near together, and so overlap, that the sounds produced when they are diseased are not easily distinguished by their position alone ; it is rather by the course which these bruits take in accordance with the laws of conduction that we are able to separate and distinguish them. Nevertheless it is as well to know the relation of the valves to one another and their exact position on the surface. The heart retains a tolerably fixed place in the chest, so that if a perpendicular line be let fall along the left edge of the sternum two thirds of the organ would be on the right side of this line and one third on the left. The base of the heart is at the third rib or higher if we reckon the appendages. The apex is in the fifth intercostal space or at the upper edge of the sixth rib, two inches below the nipple and one to its inside, the nipple being placed pretty uniformly over the junction of the fourth rib with its cartilage. The area of cardiac dulness in each direction is about two and a half inches, the lung covering the upper part of the heart leaving a triangular space having its base line running from sternum to apex. The sigmoid valves are placed over the third left costal cartilage, reaching a little above and a little below, the pulmonary at the upper edge of the cartilage, the aortic behind and a little below as well as a little nearer the median line; the mitral is found at the third intercostal space a little outside the aorta, the tricuspid is in front of the mitral and is nearer the median line behind the sternum. The aorta passes to the right, following the long axis of the heart, whilst the pulmonary artery passes to the left. Although the stethoscope cannot separate the sounds at the seat of their production when these valves are diseased, yet (owing to their taking certain directions as we shall now describe) there are special parts of the chest which we regard as the spots where particular valvular murmurs are best heard, viz. the second right costal cartilage for an aortic bruit, the second left costal cartilage for a pulmonary bruit, the fifth right costal cartilage for a tricuspid bruit, and the apex of the heart wherever situated for a mitral bruit.

Conduction of sounds.—Sounds arising in the heart are conducted by the solid walls of the organ, by the direction of the flow of blood, and by the surrounding tissues outside the organ. If we consider the heart lying deep in the chest, the left side being posterior and covered by the right, which is in front of it, we shall see how enveloped the mitral valve is and through what a mass of tissues any sound produced therein would have to pass before reaching the surface; and if, again, we consider that the valve is attached to the left ventricle, and that the latter touches the chest wall by its apex, it becomes obvious that any sounds produced in this valve would be more readily conveyed to this point than directly to the surface through the right side of the heart and the mass of tissues described. It happens, as anticipated, that all sounds produced in the mitral valve are heard best at the apex of the heart; this is consequently called the *mitral area.* Again, the sounds produced in the interior of the heart are conveyed more readily in the course in which the current runs, consequently the bruit produced in the mitral orifice by obstruction or regurgitation, although better heard at the apex than at the base for the reasons just given, would have also its direction determined in the two cases by the different course of the blood; the bruit produced by the obstruction or the direct murmur would be carried to the apex and there limited, whilst that produced by regurgitation would be carried back to the auricle and therefore heard in the dorsal region. In the case of obstruction of the aortic orifice the sound would be carried to the right along the aorta, whilst in the case of aortic regurgitation the bruit would be carried downwards. The course of this bruit is very remarkable and has received several interpretations from different authors. The blood flowing back through the imperfect aortic valves passes into the ventricle, and it might be thought that the bruit would be traceable down to the apex; this is sometimes the case, but rarely. As a rule, the bruit is carried down the middle of the chest, along the sternum, and is heard best at the lower portion of this bone just above the ensiform cartilage. Dr Balthazar Foster suggests an explanation of the different course of the bruit (if it ever is heard loudest at the apex) in the fact that different valves are affected in different cases, and that if the left sigmoid near the mitral is affected, the blood falls directly back into the ventricle and the bruit is heard best at the apex, whereas if the right or posterior sigmoid valve is diseased, the regurgitant blood impinges on the septum, and the bruit is carried down the right side of the heart. Dr Sibson attempted an explanation by supposing that the sinus arteriosus, being the shallowest part of the right ventricle, was a better conductor of sound than the left ventricle, and so a bruit passed directly through it. There seems, however, a much better and simpler explanation than either of these, and that is that the bony tissue of the sternum lying directly over the aortic valves where the bruit is produced, being a so much better conductor of sound than the fleshy heart, the bruit is more readily carried down the sternum than along the ventricle to the apex. The explanation is good and sufficient. There can be little doubt if the heart were out of the body and a sound were produced at the commencement of the aorta, and a piece of bone or wood were laid upon it and the other end placed to the ear, that the sound would be better conveyed along this material than along the fleshy substance of the ventricle.

The diagnosis, therefore, of valvular bruits depends upon their *rhythm*, *position*, and *direction*. Of the four commoner forms of disease just mentioned, an obstructed mitral orifice is known by the bruit being præsystolic and limited to the apex. A regurgitant mitral bruit is known by being systolic, heard best at the apex and conducted to the back: an obstructive aortic bruit, by being systolic, heard best at the base and carried upwards to the right; a regurgitant aortic bruit, by being diastolic, heard at the base, and carried down the sternum. If a regurgitant tricuspid bruit exists, it will be systolic and heard to the right of the ensiform cartilage.

We will now take each of these cases separately :—

Stenosis of mitral orifice with præsystolic murmur.—This murmur is heard during the diastolic period and terminates abruptly with the first sound; it is therefore styled præsystolic. It is limited to the apex, is characterised by its harshness, and is often called "churning" or "grinding," and thus unlike the soft bellows murmur. It is also very variable in intensity, differs in loudness day by day and often according to the posture of the patient. If the finger be placed on the carotid whilst the ear is over the heart the exact rhythm of the bruit may be ascertained. It is evident that if the natural first sound of the heart is due mainly to the tension of the mitral valve, it cannot be produced when the valve is converted into a fibrous ring; it is consequently short and sharp and resembles rather the second sound of the heart than the first. Herein lies one explanation of the long failure to recognise the præsystolic bruit; the lengthened murmur followed by this short click naturally suggested a systolic murmur followed by the second sound, whereas the click was the modified first sound preceded by a bruit occurring during what in health is called the period of silence. The mistake was further strengthened by the almost total absence of the second sound at the apex; this being diminished in intensity partly from being lost in the long harsh bruit preceding it, and partly from the smaller amount of blood sent into the aorta. It may, however, be heard well at the base and may even be accentuated as in mitral regurgitation. Another difficulty arose in accepting the existence of a direct mitral murmur from the fact that in health no sound is produced during the passage of blood through the auriculo-ventricular orifice and also from the fact that the contraction of the auricle is momentary, whereas the alleged præsystolic murmur is both long and harsh. The answer has been that the conditions in disease are altered and the auricle being abnormally thickened and its action slow, an abnormal bruit may be produced by its contraction. It is also possible that the præsystolic bruit may be in part produced by the blood passing into the ventricle prior to the auricle's contraction, seeing that the auricle and its veins must exert considerable and very abnormal pressure upon it. The cardiographic tracing also which gives in health a slight elevation of the lever corresponding to the systole of the auricle, produces in these cases a long and elevated curve.

Another very frequent sign of a narrowed mitral orifice was pointed out by Laennec and confirmed by all subsequent writers—a tremor or *frémissement* felt when the hand is placed over the heart; it is curious how this observation did not long ago lead to the knowledge of the bruit under these circumstances being not systolic, for if the heart beats slowly the thrill is distinctly felt during the recession of the apex and not during the impulse. It is very remarkable what an insight Laennec had into the form of disease of which we are speaking. He illustrates his remarks by the case of a young man in whom he says "the hand placed over the region of the heart feels the pulsation strongly and accompanied by the purring vibration; the stethoscope applied between the cartilages of the fifth and seventh ribs gives the following results:—Contraction of the auricle extremely prolonged, accompanied by a dull but strong sound exactly like that produced by a file on wood. This sound is attended by a vibration sensible to the ear and which is evidently the same as that felt by the hand. Succeeding this a louder sound and a shock synchronous with the pulse point out the contraction of the ventricle, which occupies only one fourth part of the time. From these signs the following diagnosis was given:—Ossification of the mitral valve, slight hypertrophy of the left ventricle." The immediate followers of Laennec taught the same. For example, Hope writing in 1832, says, "When the mitral valve is contracted, a murmur accompanies and sometimes entirely supersedes the second sound, being occasioned by the obstructed passage of the blood from the auricle into the ventricle during the diastole of the latter."

Another frequent sign of mitral stenosis is reduplication of the sounds of the heart. It is obvious that if the two sides of the heart did not act together four sounds might be heard instead of two. If, however, this want of synchronism occurred, it is still doubtful whether the two first sounds ever could be separated into two, so that double sounds are probably always due to the second being heard twice. In the case under discussion the heart's action is thrown out of gear by the hypertrophy of the left auricle and by the increased pressure on the right side of the heart. Under these circumstances, the two ventricles not acting together, the aorta and pulmonary artery would be thrown out of order and a double sound would be produced by the closure of the sigmoids. Dr George Johnson has suggested that one of these sounds may be not valvular but due to the auricular systole. Dr Sansom also thinks it may occur from a tension of the mitral valve at the time when the blood is thrown back during the diastole of the ventricle and therefore is not a normal second sound. This double beat, or, when combined with the first, this triple beat of the heart, is sometimes heard under other conditions of impediment to the circulation, but it is most common in the case of mitral stenosis. It is called by the French *bruit de rappel*, and is like a hammer rebounding on the anvil.

For certain reasons the contraction of the auricle is sometimes not equal to the production of a bruit in stenosis and then the case is characterised by the almost total absence of the first sound. This may arise from weakness of the auricle or from less pressure being exerted on it from behind. A *frémissement* might still be felt, since a less number of vibrations than would produce a sound would be felt by the hand. In some cases, although the mitral orifice is narrowed, the valve is so rigid that blood regurgitates through it; under these circumstances a systolic bruit exists as well as a præsystolic; the two may be distinctly separated, though they sometimes run together. As has been before remarked, we occasionally find cases of stenosis which have occurred in childhood and which have directly influenced the nutrition of the heart by diminishing the amount of blood circulating through the body; the size of the mitral orifice being a measure of this amount. The heart has consequently been much diminished in size. The pulse is small and sometimes regular, showing no characteristic tracing by the sphygmograph. Occasionally when the double beat is present, every systole of the ventricle does not fill the aorta and the pulse is less frequent than the heart's action.

Stenosis of tricuspid with præsystolic murmur.—This is occasionally met with; the bruit being heard best over the fifth right costal cartilage near the sternum. When after a time the blood is thrown back on the lungs and venous system, then all the other ulterior consequences take place as in the case of regurgitation. Dr Bedford Fenwick has collected forty-six cases, the majority of which were in young women ('Path. Trans.,' 1881, p. 48). In most of them there coexisted disease of the mitral valve, and in half there was no rheumatic history giving a clue to their origin.

Imperfection of mitral valve causing regurgitation and systolic bruit.—The bruit is of a blowing character, heard loudest in front over the apex region, heard also along the side of the chest under the axilla and in the back over the angle of the scapula. Owing to the backward obstruction in the lungs and the right side of the heart the blood during the contraction of the pulmonary artery recoils with more violence on the sigmoid valves, and consequently they contract with greater force; this produces an accentuation of the second sound heard at the base towards the left side. The apex-beat may be slightly lower than natural and the pulse in extreme cases is small and very irregular. The other physical conditions are mainly due to the engorgements of the venous system from the blood being continually held back in the vessels. Thus the veins in the neck which are not usually observable are seen markedly to stand out, especially on the right side, where they can often also be seen pulsating. This distension sometimes arises from the impulse given by the carotids; in other cases it is due to the contraction of the auricle, and in others to a real regurgitation through the tricuspid. It is usually the external jugular which becomes prominent, although the internal may also be sometimes seen. The valves are clearly visible and become more so on coughing. The engorged right side of the heart may be felt beating in the epigastrium, and if one hand be placed in this region and the other in the right loin, the whole liver lying between the two hands may sometimes be felt pulsating (Dr. F. Taylor, 'Guy's Hosp. Rep.,' 1875). The engorgement of the veins is shown by the enlargement of the liver and by the dropsy, also by the hæmoptysis and albumen in the urine.

Regurgitation through tricuspid with systolic murmur.—If it be true that in a gorged heart reflex often occurs through the tricuspid, and if, as we know, this is not accompanied by a bruit, we may safely assert that no sound is produced by regurgitation through a healthy valve. When a bruit does exist it is usually in very chronic cases of bronchitis, and where the valve has become thickened and intensified, probably on account of its abnormally increased action.

Obstruction of aortic orifice and systolic bruit.—This generally arises when there is some roughness of the valves or disease of the aorta; a simple stenosis without regurgitation not being very common. The murmur is heard at the base of the heart and is carried across the sternum to the second right costal cartilage. The natural first sound heard at the apex is not quite so good as in the healthy heart and is partly covered by the bruit here faintly heard; the second sound which is produced by the closure of the sigmoid valves is indistinct. The heart is hypertrophied and the apex-beat is observed to be slower than natural and more external. The pulse is small, slow, long, and regular.

Imperfection of aortic valves with diastolic bruit.—We have already spoken of this bruit produced at the orifice of the aorta and carried down the sternum. The imperfect valve or valves usually causes a murmur as the blood passes over it into the aorta, and thus, as a rule, in cases of diseased aortic valves a double aortic murmur is present; or, as it is sometimes called a "see-saw murmur." The two bruits may differ in intensity, which is of the utmost importance in a diagnostic point of view; the louder the first the more favourable the case, and the louder the second the converse; although if the latter be prolonged it does not show so much disease as when the bruit is very short and the natural sounds altogether obscured; for the latter often denotes that the blood is falling back with great facility into the ventricle. The bruit is sometimes musical, and occasionally can be heard at some distance from the patient. Sometimes also a thrill accompanying the bruit can be felt by the hand. The heart in these cases is much enlarged, the apex-beat being felt in the sixth or seventh space, and the area of dulness is increased in all directions, and in young persons an actual projection of the chest may occur. On account of the failure of support to the current of blood in the aorta and of the enlargement of the ventricle, the pulse is remarkable and characteristic. It has two well-marked features. First of all a large quantity of blood is sent out into the distended vessels and the pulse is therefore regular, large, and visible to the eye; and, secondly, this large full pulse suddenly collapses under the finger. The reason is that when this large amount of blood is sent into the aorta and the latter contracts, the valves behind, not being competent to support the column of blood, the pulse is not sustained and we are cognisant, on placing the finger over the artery, of this collapse. This pulse has many designations: Corrigan's, the water-hammer, splashing, collapsing, receding, jerking, locomotive, kicking or shuttle pulse. If a novice, with no intention of feeling the pulse, grasp the wrist of a person suffering with aortic disease, he at once becomes aware of its peculiarity by the remarkable throbbing. Dr Galabin says it is the suddenness or sudden blow which renders the pulse characteristic. As there is a want of relation between the amount of blood and its vessel, a very similar pulse is felt in cases of hæmorrhage, and therefore "the pulse of unfilled arteries" is a name sometimes given to it. As might be supposed, the jerk would be increased when the limbs are raised, and thus if the arm be held up the character of the pulse is much exaggerated. The pulsation is also felt in the small arteries, as the digital, where it is not usually detected, and the pulsation may also be readily discerned in parts which are highly vascular. Thus, the retinal vessels may be seen to pulsate, and any patch of capillaries on the skin may be seen to change colour at every beat of the heart. If an artificial blush be made with the nail on the skin and this be watched it may be seen to grow red and pale with every pulsation; this is known as the "capillary pulse." Sometimes the pulsation is propagated to the veins beyond when they also may be seen to beat. The systolic bruit is carried very often a considerable distance along the vessels and sometimes the diastolic also. In the femoral artery, for example, a double bruit may sometimes be heard. If not, it may very frequently be produced by using pressure with the stethoscope. In all probability, if a diastolic bruit be easily brought out in this manner, regurgitation must be occurring in the larger vessels.

It may be here remarked that all the signs just mentioned denote incompetency of the aortic valves; it does not follow, however, that they are diseased, as their want of closure may arise from dilatation of the ascending aorta or disease of its coats. For example, a yielding of the sinuses of Valsalva just above the aortic valves has been proved to interfere with their perfect action. Dr Flint, of New York, maintains that a præsystolic murmur may sometimes be heard in cases of aortic regurgitation. He believes it is due to the vibration of the mitral segments even when the valve is inactive, owing to the over-engorgement of the ventricle from the reflux of blood into it from the aorta.

As regards the *relative importance* of the valvular diseases, probably that of aortic obstruction is the least important, next to this stenosis of the mitral valve, which with a compensating hypertrophied auricle may endure for years. Regurgitant diseases are far more serious, but mitral regurgitation less so than aortic, the latter being a condition often leading to sudden death. This opinion is corroborated by Dr Peacock and Dr Bristowe, but there are authors who regard mitral stenosis as graver than mitral incompetence.

These valvular diseases, however, are all less important than those which commence in the cavities themselves, for dilatation of the heart, fatty and fibroid degenerations, give rise to the most urgent cardiac symptoms, often run a rapid course, and not unfrequently terminate in sudden death. To these we will now draw attention.

Before speaking of these primary affections of the cavities of the heart it is important to remember the changes which take place in them in connection with valvular diseases, and may be often considered compensatory or conservative; for it is evident that as long as these compensations hold good, the primary fault in the mechanism may be counterbalanced, and therefore it is the failure in these secondary beneficial alterations which mainly contributes to all the distressing consequences of heart disease. The enlargement and hypertrophy of the several cavities in valvular disease overcome the imperfections of the valves which would otherwise be at once felt, but as soon as these compensatory conditions fail, then the symptoms appear with increased force. Each particular case must be considered before maintaining the universal truth of this. Should the ventricle remain tolerably small in mitral regurgitation, it might perhaps be more effective than if it hypertrophied and sent back the blood on the lungs. But it may be said generally that as long as hypertrophy of the ventricles is equal to propelling the blood through the systemic vessels and lungs, the organism works fairly well, but should the ventricles become over-dilated, or thinned, or the muscle degenerate, then cardiac symptoms at once arise.

The state of the cavity therefore and of the muscle is of equal or of more importance in considering the power of the central organ of circulation than that of the valves. It follows too that if the cavities themselves should from any cause be the seat of primary disease the very worst form of cardiac affection is the result. This is constantly seen to be the case, and therefore to consider heart-disease as an affection of the valves only is to regard it in a much too partial manner. In advanced age a large proportion of the cases of disease of the heart are those of the muscular tissue only, and if any incompetence of the valves be found this may have occurred as a secondary consequence. Again, in persons excessively fat, the organ may become overloaded with adipose tissue both on the right and left side, rendering the ventricles unequal to their work; or, what is more usual in persons advancing in life, the muscular tissue throughout becomes fatty or soft and unable to propel its contents, or in consequence of the weakness of the tissue the ventricle dilates; this prevents the due closure of the mitral valve; regurgitation may take place through it, a bruit is developed, and all the symptoms of mitral incompetency ensue. This is probably one of the commonest causes of mitral regurgitation. When the heart is examined after death, although no structural disease of the valve is seen, it is very obvious that with a largely distended ventricle the valves could not have closed. These changes in the cavity may come about in various ways; it may become fatty and degenerate in intemperate persons or sometimes in the gouty. Dilatation without this may occur sometimes after severe illnesses, and Dr Goodhart believes this is often the case in scarlatina. It no doubt occurs in rheumatism without inflammation of the serous surfaces, and it may arise after excessive hæmorrhages. In rheumatism, as before said, a real myocarditis may sometimes occur, and its results be seen in fibroid changes in the muscular tissue of the heart many years afterwards. Herein is a very striking example of disease of the heart originating in the muscular substance. The various symptoms and physical signs accompanying these enlargements have already been dwelt upon (vol. ii, p. 23).

It is in these secondary valvular diseases of the heart that the origin of bruits and abnormal sounds has been so much discussed, and it is their investigation which has been mainly instrumental in uprooting the old theory that bruits depend upon the friction set up by a diseased valve. According to the more recent explanation, that they are due to the movement of a number of currents when the blood passes from a smaller into a larger space, it is clear that they may arise under a number of circumstances where no cause of friction obtains. We can see how a want of relation between the vessel and the blood, as where a vessel is not only dilated, but the fluid within scanty and poor, can produce a tumult among the particles and a bruit result. In anæmia such a bruit is heard at the base of the heart and directed generally towards the left side in the direction of the pulmonary artery; it is also heard in the carotids and as a continuous murmur in the jugular veins. Another explanation of the anæmic bruit heard so often in chlorosis has been given by Naunyn, and is strongly upheld in this country by Dr Balfour, that it is a valvular murmur and due to regurgitation through the mitral valve, that the ventricle fails fully to contract, that the mitral does not close, and the blood, being thrown back into the auricle, causes a bruit to be heard at the base over the auricular appendix of the left auricle. The theory is not generally accepted for the reason that the auscultatory phenomena do not agree with those which all admit exist in undoubted mitral regurgitation. Should, however, the theory not hold good in this case it is probable that there are temporary and functional relaxations of the mitral valve, by which for a time it ceases to be efficient and yet after a time recovers itself as the blood improves in quality and the tissue in strength. But even with these explanations many bruits exist whose origin has not been made clear; for example, the very common case of endocarditis evidenced by vegetations on the mitral valve is accompanied by a bruit the cause of which is not at all obvious. It may safely be asserted, since the first sound of the heart is due mainly to the stretching and closure of the mitral valve, the causes of the natural sound must be seriously interfered with when this valve is thickened or covered with vegetations; but this scarcely explains the existence of the loud *bruit de souffle* which is so often heard in chorea. It has been suggested that if in such a case a regurgitation did occur, the inactivity of the valve might be explained on the supposition that, being loaded with vegetations, its specific gravity would be altered and therefore would not float up in the ordinary natural way. In other cases where a temporary dilatation of the ventricle may be supposed to exist it has been thought that the blood, circling in eddies behind the curtains of the valve, might produce abnormal sounds.

There are still other bruits which have not yet received a full explanation; for example, a bruit heard under the clavicle, more especially on the left side and occurring intermittently. This in some cases is obviously in the subclavian artery and has been thought to be due to muscular pressure on the vessel. In some cases where there is a suspicion of phthisis it has been thought that a compression of the pulmonary artery by a consolidated lung may have caused it.

Occasionally bruits may be heard which apparently depend upon movements of parts altogether outside the heart, and these are called *exocardial.* They depend sometimes upon adhesions between the sac of the pericardium and the pleura, which becomes stretched during the heart's action. They are clearly outside the heart when they alter or cease with the respiration; for example, altogether disappearing when the patient holds his breath in the extreme condition of expiration or inspiration. It has been suggested that when these intermittent sounds resemble bellows murmurs the heart may be beating against an isolated tongue of lung and that at every stroke a portion of air is driven out of it.

Besides the more ordinary murmurs arising from well-known causes which we have been describing, we occasionally meet with others which have a special origin, and these require all our acumen to unravel; for example, a bruit might be caused by a communication between the aorta and pulmonary artery owing to the rupture of an aneurysm, and the sound be peculiar both in character and rhythm, as perhaps a diastolic murmur at the base of the heart. Then also the bruits of circumscribed aneurysms might be heard at any part of the chest, and finally bruits arising from the various malformations of the heart. One of the commonest of these malformations is that in which the aorta and pulmonary artery come off together from the ventricles or are in some other way united; a loud systolic bruit is then heard all over the chest, especially at the base, and unlike ordinary valvular murmurs is often carried upwards. When carried to the left clavicle such a bruit generally denotes a congenital narrowing or partial closure of the pulmonary artery. It is very remarkable that in these latter cases the patients have generally died of phthisis.

The pulse.—This is the wave produced when the blood is driven into the elastic arteries, and they contract again upon it. Many of the peculiarities connected with it have already been dwelt upon, so that we shall merely state concisely its principal characters. It only requires a moment's consideration to see what a large number of elements are in operation to produce a particular character of the pulse at any given time, and therefore what variations it is liable to undergo both in health and disease. If the heart and vessels be regarded as a piece of mechanism, with its valves like a bag and pipes of exit, it will be evident that the pulse must vary with the force and regularity with which the blood is driven out of the impelling organ; it must be affected also by any derangement of the valvular apparatus which close its openings; it will vary also according to the nature of the pipes or vessels through which the fluid flows, viz. their elasticity or rigidity. Since, too, the blood has to be driven not only into vessels, but into the tissues beyond, the readiness with which the latter receive the circulating fluid must necessarily affect the flow and impress itself upon the pulse. Then moreover the character of the blood itself must be taken into account, since fluids of different qualities circulate with different amounts of freedom, and finally as the coats of the vessels are living and their firmness influenced through the nerves as well as the heart, the state of the nervous system must be noted as affecting the pulse in a very marked degree. It is therefore obvious that the movement of the blood through vessels or the pulse is most complex and apt to be influenced by a large number of causes. If you repeat the experiment performed in Guy's Hospital many years ago by Mr Wilkinson King, that of attaching a bristle to the skin over an artery by means of a piece of wax, you will at once perceive that the pulse is something more than an up and down movement, but that the end of the bristle is making vibrations or curves. If this bristle, which Mr King called the "sphygmometer," could be made to take tracings, you would then see the natural movements of the pulse.

The first thing observable in a pulse is its rapidity or number; this, except under peculiar circumstances, is due to the rate at which the heart is working. If the number be great we usually speak of a *quick* pulse, but most writers have endeavoured to limit the term quick to the rapidity of the beat or systole of the heart, and using the word *frequency* for the number of pulsations. The force also of the pulse depends in great measure on the heart, and the same may be said of its regularity. The terms *large* and *small* speak for themselves. The large, full, distended vessel is readily distinguished from the small and contracted one. Fulness, however, does not necessarily correspond with relaxation nor does smallness with contraction. In the case of imperfect aortic valves or in hæmorrhage, fulness may be due to a relaxed vessel, and firmness or tension of the vessels implying a contraction of the coats to various influences. The older writers used to speak of a *quick, full, and hard* pulse of some kind of inflammations as pneumonic, and of a *hard and wiry pulse* of other inflammations, as of the peritoneum. In the latter case the influence of the sympathetic nerve on the heart and vessels produced its characteristics. The tension of the vessel has been long observed as a peculiar quality of renal disease, and it is well exemplified in the tracing made by the sphygmograph. It is important to distinguish between this hardness produced by a temporary contraction of the coats of the vessels from an actual hypertrophy or thickening of their walls. In Bright's disease they both exist, the former preceding the latter. Nerve causes, especially those originating in the brain, operate powerfully in slowing the action of the heart, the slow labouring pulse of brain disease being universally recognised. Then besides the actual force and varied action of the heart and arteries, and the influence exerted over them by the nervous system, the state of the blood itself will affect them according to its poverty or otherwise. Then the freedom with which the blood is delivered into the tissue will react on the blood-vessels and affect the pulse; one occasional result of any impediment to a free circulation is the production of another beat in the vessel constituting the so-called *dicrotic pulse*. Various mechanical derangements of the valves necessarily affect the pulse, as we have already seen in speaking of these different lesions. When one or two beats are left out the heart is said to be intermittent. Intermittence and irregularity have already been considered in connection with functional affections (*supra*, vol. ii, p. 3). It may be observed that irregularity of the pulse is sometimes its normal condition; in a case where this was unknown by the medical attendant during the final illness of the patient it was regarded as an important symptom, but the heart was found perfectly healthy after death.

The *sphygmograph* consists essentially of a needle which is moved by the pulsating artery, while its vibrating end is made to trace itself on paper. The pulse, as felt by the finger, shows itself in such a tracing as one curve followed by another, and sometimes by a third. The second wave is occasionally felt by the finger, and if so the pulse is called "dicrotic;" the tracing needle, however, shows its existence in all cases. These waves or pulsations are very similar to those which are felt by the hand or foot when placed on an elastic pipe through which water is being pumped. It is the first wave and its size that measures the force of the pulse; when large, it shows strength of the heart and distension of the vessel; when wanting, it shows a weak heart or a relaxed vessel, as in aortic valvular disease, where the column of blood is not sustained by the valves.

If, however, we look at a sphygmographic tracing we see yet another stroke preceding the curves of which we have been speaking. This is generally perpendicular, and reaches high above the following curves. It is still a question whether this constitutes any part of the tracing, whether, indeed, there is anything in the pulse to produce such a stroke. It is nearly perpendicular, or sometimes when the heart's action is very quick will slope the contrary way. Some of the best investigators regard it as a mere jerking up of the lever at the moment when the blood enters the aorta, and therefore anterior to the true pulse. This seems to be Dr Galabin's opinion in most cases.* If it is really caused by the pulsation of the vessel, it is so intimately blended with the curve which follows that they cannot be separated by the sensation conveyed to the finger. This upright line is called the "percussion stroke."

The first curve or wave which follows constitutes the most important part of the pulse; this it is which is mainly felt by the finger and by which we judge of its strength and the tension of the artery. It is called the *tidal wave;* the end of this curve corresponds with the fall of the vessel and the closure of the aortic valves, and is therefore called the *aortic notch.* The next curve is only occasionally felt by the finger; when so the pulse has been long called dicrotic, this is therefore the *dicrotic wave.*

The first or tidal wave best measures the strength of the pulse. Dr Mahomed says its size may be found by drawing a line from the apex of the percussion stroke to the aortic notch; if it is much outside this, great tension is denoted. It is clear that if there be little tension the tidal curve will disappear and the pulse be marked by a simple falling line; and if this line fall very low (which would be denoted by a low aortic notch) the vessel is relaxed, as in aortic valvular disease. If the line of the first curve should fall very low towards the notch which indicates the closure of the aortic valves, and then another rebound occur, so as to produce a second large wave, we have in the tracing exaggerated dicrotic characters; and if the lever fall still lower, so as to reach below the basal line and then rise to produce the dicrotic wave, we have what is called a *hyperdicrotic* tracing. We see, then, that the larger the tidal wave and the higher the aortic notch from the base line, the greater the tension of the vessel and hardness of the pulse, whilst on the other hand, the nearer this notch approaches to the line the lower is the arterial pressure. It must be remembered that the finger can detect the hardness or contracted state of the vessel during its diastole as well as systole, although the sphygmograph cannot.

The sphygmograph shows the rhythm and number of pulsations. It exhibits well the hardness of the pulse by the high and raised tidal wave; also a soft pulse by the absence of this; a dicrotic pulse by the marked size of the second wave. Therefore in Bright's disease, aortic disease, and in fevers, the tracing is characteristic. In mitral regurgitation, with the weak ventricle and diminished discharge of blood, the tracing is a mere irregular curved line; in mitral stenosis there would be nothing characteristic. In aneurysm the sphygmograph is often of great service, for if the subclavian artery be given off from the sac the tension is much decreased and the pulse on that side may be much smaller and feebler than the other, but sometimes only to an extent that the sphygmograph alone can measure. Dr Galabin says that one of the most characteristic features of the tracing in aortic regurgitation is the extreme amplitude of the percussion wave and the flatness of the diastolic portion. The latter corresponds with the emptiness of the artery during the diastolic period.*

Symptoms.—These may be subjective and objective, or those which affect the feelings of the patient and cause him to seek medical relief, and those which are only discoverable by the physician. It has already been said that so long as the hypertrophy of the heart overcomes the imperfection caused by the valves, no disturbance in the circulation need arise, and consequently no unpleasant symptoms be experienced; but immediately the cavities become unequal to the increased labour thrown upon them, all the symptoms denoting cardiac disease ensue. Should the imperfection in these cavities be a primary one owing to degenerations in the muscular tissue, it is clear that we have present from the commencement one of the worst forms of heart disease. Under these circumstances, whether the weakness of the heart is a primary or secondary affection, various distressing symptoms ensue, arising from the impeded and disturbed circulation, the most striking being the engorgement of the venous system, together with the various changes in the organs before noticed, including dropsy. In the treatment of heart disease, therefore, our efforts should be especially directed to strengthen as far as possible the weakened heart, and then to relieve the symptoms induced by the overloaded blood-vessels.

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* [See Dr Galabin's papers in the 'Guy's Reports,' 3rd series, vols. xix and xx.—Ed.]

As regards the several symptoms, we will mention first the subjective, and the most important of these is *cardiac apnœa.* The patient becomes breathless upon increased exertion, so that walking up-hill, upstairs, or dressing, particularly distresses him. He is seen to pant, but has not dyspnœa in the true sense of the term, for he has no difficulty in filling his chest with air. It would seem that the blood not being pumped through the lungs with regularity, the relation between the respiration and circulation is consequently lost and the breathing becomes irregular; the patient has to stop breathing for a moment in order to recover the proper rhythm. If a patient describe to you such symptoms, and if they can be looked upon as genuine, no doubt can exist as to the fact of the heart being weak or diseased, even if it cannot be proved by physical signs.

Another symptom which patients sometimes complain of is *palpitation.* This is by no means so important as the apnœa, since it is a symptom so very commonly produced by nervous and other causes acting on the heart from without which have been discussed before (vol, ii, p. 6). Palpitation is altogether subjective and is rarely felt except the heart beats intermittingly. Now, in some of the worst forms of cardiac disease the action is slow and regular. Then nothing is perceptible to the patient; and even when the heart is very irregular, there need be no consciousness of it. It is more especially in the intermittency arising from functional disturbance that the patient is conscious of a beating or fluttering, and seeks advice for what he believes to be "heart complaint." Probably if all the cases in which a patient complains of palpitation were put together it would be found that in a very large majority of them there was no disease of any kind and that the patient was nervous and dyspeptic. It is worthy of remembrance, however, in forming a diagnosis in such cases that although marked dyspepsia and flatulence may be found to be the causes of the patient complaining of palpitation, yet these gastric symptoms are very frequent concomitants of heart disease. They are observed especially after a meal and very frequently on the patient lying down at night, when constant eructations take place from the stomach; the case might then be regarded as one of gastric disease when it is really cardiac. The relations of the stomach and heart through the pneumogastric nerve are very intimate, and thus we can understand how one organ participates in the weaknesses and disturbances of the other. The dyspnœa being greater when the patient lies down is probably due to mechanical causes, the pressure of the stomach and diaphragm. This is one reason why he prefers to sit all night in a chair with his head leaning forward.

The patient with heart disease after describing his symptoms of breathlessness, of palpitation, or of dyspepsia seldom fails also to dilate upon his disturbed and *sleepless nights.* He says as soon as he falls off he starts up in fear of suffocation. There are many reasons for this; as the heart requires the largest expanse of lung for the circulation, so the best position for the patient is the dorsal one with the head raised, a position to which he had previously been quite unaccustomed, and therefore incompatible with the usual rest; then again for the production of sleep a quiet regular circulation through the brain is necessary, but this is often impossible when a heart is beating quick with various rates of force. Then besides these there is another cause, probably the most important, which seems to be due to some action on the medulla oblongata rendering it less susceptible of its function in carrying on the respiration; for although it is said that the spinal system never sleeps, the statement is true only in a sense; during ordinary sleep the deep breathing certainly indicates a change in the respiratory process. In disease of the heart this change, probably due to an altered vascular state of the medulla, immediately produces suffocative symptoms and awakens the patient. A peculiar form of respiration and which may generally be regarded as a fatal sign is the Cheyne-Stokes respiration, or the ascending and descending respiration—marked by a series of respirations becoming shallower and shallower until it altogether ceases—then after a long pause the series begins again.

These are the chief symptoms complained of, but before long the disturbed circulation gives rise to positive objective signs in all parts of the body. In mitral disease, in a certain number of aortic cases which have advanced to the last stage and in various cases of primary affections of the heart tissue, the venous circulation becomes obstructed and the changes already described ensue in the various organs of the body. The cardiac apnœa becomes aggravated by an actual *congestion of the lungs:* the patient has a cough, and begins to expectorate mucus tinged with blood, and sometimes enough comes up to be called hæmoptysis. On examining the patient, besides discovering mucous râles, we find that the lower part of the chest is imperfectly filled with air and perhaps there is dulness on percussion at the base of both lungs; as a rule it is the lower lobe of the right lung which first becomes blocked. No satisfactory explanation of this engorgement of the right side has yet been forthcoming. At this time the lung is in the condition known as brown induration or splenization, and if blood has been expectorated there are probably effusions of blood in its tissue; the coloured mucus implies an intense congestion of the bronchial membrane also. If the lungs be gorged in this manner the whole venous system is necessarily much obstructed, *dropsy* occurs in the legs and in the abdomen, and the veins are seen to be much enlarged in the neck. The skin of the upper part of the body is also yellow; this is due to engorgement of the *liver*, which is in the state called nutmeg (p. 50); the liver is felt much enlarged, reaching to the umbilicus, and, if the hand be placed upon it before and behind, may sometimes be felt to pulsate. This enlargement of the liver is often a great help to diagnosis. A medical man may be called to a patient for the first time, whom he finds dropsical, with albumen in the urine and a state of heart which, from its weakness and the sounds of bronchitis, is not at once easy to make out. He may be discussing in his own mind the question whether cardiac or renal disease is the most appropriate name to give to the case, but if the liver be enlarged he at once decides in favour of the former, as no kidney affection would cause its enlargement. It is therefore a great aid to diagnosis. The *kidney* also becomes gorged with blood, and the urine scanty, of high specific gravity, and albuminous; after a time casts may be found, from an actual tubular nephritis being set up in the kidney, just as a bronchitis is produced in the lung. It is important in this case also to discover, if possible, whether the albumen denote a substantive disease of the kidney or a mere congestion, for the prognosis may turn upon the answer. Urine of the nature I have spoken of arises from congestion and it does not indicate of necessity any irrecoverable disease of the organ, and yet it is a serious complication, since the impaired action of the kidney aggravates the dropsy. Very often the discovery of heart disease and kidney disease combined seems to have taken away all hope of recovery or even temporary improvement, and yet it is this very combination which may suggest a reason for hopefulness. For example, a patient with cardiac disease has, owing to cold or some unknown cause, an aggravation of his symptoms, and the kidneys become congested, they fail to accomplish their due amount of work, and the dropsy, together with all the other symptoms, become quickly aggravated; with this double affection the case seems to be desperate, when by treatment and time the kidneys recover themselves, the albumen ceases to appear, the urine begins to flow in natural quantities, the dropsy departs, and the patient once more returns to comparative health. Congestion of other organs also occurs in heart disease, and the extreme redness of the *stomach* often seen after death is no doubt incompatible with its proper function and therefore one cause of sickness and want of appetite. As regards the *dropsy*, this commences in the legs, then passes up until it reaches the peritoneum and afterwards the pleura and the pericardium. Why the fluid should fill one pleura and not the other is not always evident, but it may be due to mere accidental causes, as, for example, to a closely adherent lung on one side. It is said, and probably with some truth, that all pleuritic effusions, both active and passive, affect the left side more than the right. The skin in dropsy often becomes tense, shiny, and red. It may proceed to actual inflammation, including the cellular tissue beneath. Bright observed that inflammation and sloughing were more likely to occur when the kidneys were involved, probably from the serum containing irritating urinary ingredients.

The symptoms above described arise from venous engorgement: they are more especially seen in cases of mitral imperfection, whether arising from primary disease of the valve or dilatation of the ventricle. In aortic disease, while the force of the ventricle is equal to overcome the difficulty produced by the constant reflux of blood, none of these symptoms are seen; but as soon as the ventricle fails in this respect and the mitral valve is unable to close, all the symptoms of mitral disease ensue; at the same time a mitral regurgitant murmur may become audible in addition to the aortic which before existed. Before this occurs, however, the difference between the case of aortic disease and that of mitral disease is most marked; in the latter the venous obstruction produces congestion and lividity of the face, with a general bloatedness of the body, or actual dropsy; whereas in the former, owing to a small amount of blood being retained in the tissues, they become impoverished, and the patient is universally wasted. Whilst he remains quiet, and therefore no stress is put upon the circulation, his appearance might be compared with that seen in wasting disease; in fact he might at first glance be considered to be suffering from phthisis.

There are a class of symptoms observed in disease of the aorta and its valves not seen in mitral disease; these symptoms resemble those of angina pectoris. Patients are seized with intense pain in the chest combined with a sense of constriction, and this pain passes across the chest and down the arms; in these paroxysms they sometimes die. They are relieved by nitrite of amyl, by nitro-glycerine, and other remedies which are useful in angina. Sudden death is common in aortic disease, more rare in mitral.

Treatment.—This is of two kinds or has two objects, the one directed against the perverted action of the heart and the other against the effects of the obstructed circulation. For the former great object we have in our hands one of the most remarkable medicines in the Pharmacopœia, one which if given opportunely may truly snatch the patient from the jaws of death. He may, for example, be the subject of mitral disease and of all its attendant consequences, and may be approaching his end, when digitalis—for this is the remedy referred to—is administered; the heart becomes quiet, contracts with more energy, and with this improved action, all the symptoms depart and the patient is restored to comparative health. There is no such striking effect seen from the influence of any other drug. A pill consisting of digitalis, squill, and mercury had long been known as a most valuable medicine in cardiac dropsy, but formerly its administrators scarcely understood that it had any other than a diuretic action; it is evident, however, that the digitalis was influencing the heart in a very direct and specific manner, and in this way its action was beneficial. With the knowledge, therefore, which we now have, digitalis is given alone and in larger doses in those cases where its specific action is immediately wanted. Digitalis gives tone and regularity to the heart, it has been called therefore the tonic and opiate of the heart. The fact is known not only by clinical experience but by direct observation on animals. If the chest of an animal be exposed and digitalis administered, the organ is seen to contract more firmly until becoming tighter and tighter and its action slower and slower, it ceases to beat in the most complete systole. If the animal be previously poisoned by any substance which should relax the heart and stop its action during the time of dilatation, the effect of digitalis is even then more striking, the drug excites it to contraction afresh, which goes on increasing in vigour until it ceases in systole as before. Now, if in disease we have exactly the counterpart of a heart in this weak state, we have also the same valuable agent at hand. Should a patient have a very irregular and weak action of the heart, digitalis is the appropriate medicine, and since this weak action is usually a concomitant of a dilated ventricle and diseased mitral valve, it is more especially in cases where we have a systolic mitral bruit and irregular heart that we give digitalis. When patients are able to walk about, but are breathless on any increased exertion, and the action of the heart is weak, irregular, and attended with a mitral bruit—digitalis is the remedy. We should begin with ten drops of the tincture three or four times a day for a few days, and then reduce the quantity. A favourite and good form is the combination of digitalis with ammonia and senega; the latter two remedies were highly praised by the late Dr Barlow as supporters of the heart's power. Subsequently, with the smaller amount of digitalis, iron may be beneficially given; and when the digitalis has had its effect, the iron alone or combined with ammonia. Patients with heart disease may have their lives prolonged for many years by the judicious use of these two remedies. Digitalis may even further act with advantage, for if its effect be to preserve the quiet action of the heart and give it tone, its long continued use may produce a permanently tonic state in a heart which had been previously weak. Therefore, besides its immediate use as a sedative to the heart, it acts as a tonic when long continued; and many cases might be quoted where small doses given for months together have been attended by the most beneficial results. The mode in which digitalis quiets the heart is by prolonging the diastole; in cases where the organ is too quickly contracting and fluttering, this drug controls its action until the ventricle is quite filled and the systole takes place. A patient, aged fifty, having a mitral bruit, a very irregular pulse, ascites and anasarca of legs, was given digitalis in full doses until all the symptoms were relieved; he was then ordered a mixture containing the tincture to the amount of seven and a half minims three times a day. Finding the medicine useful he continued it without intermission for seven months. When seen at that time his heart was beating slowly and deliberately at the rate of 54 per minute; the remarkable point observable was the prolonged diastole. In cases where this drug has not been given at the onset and where dropsy and all the other symptoms of obstruction have set in, such as congested lung, enlarged liver, and albuminuria, digitalis may even then be given with the same striking results as those just mentioned, and together with the steadying and strengthening of the heart's action, all the engorgements and dropsy may at the same time disappear.

If the heart be not very irregular and feeble, and therefore affords less indications for the free use of digitalis, the diuretic pill before mentioned, composed of mercury, squill, and digitalis may be substituted for the simple drug. The combination is valuable for its diuretic effect, although no doubt much of its efficacy lay in the power of digitalis over the heart, a fact unknown to the first compounder of it. Other remedies which act as diuretics are also useful in cardiac dropsy, that is, always supposing the kidneys to be healthy: these are for the most part salines, such as the cream of tartar drink. The resin of copaiba also sometimes acts as a remarkable diuretic. Caffein has been recommended as a good diuretic, but the effects are very doubtful.*

Purging is also useful, and compound jalap powder administered two or three times a week will sometimes give more relief than any other remedy.

Occasionally, however, the engorgement of the lung will come on so quickly, and the whole venous system with the right side of the heart be so blocked, that venæsection may be practised with the greatest benefit; the indications for this treatment are orthopnœa, lividity of countenance, great distress of breathing, and engorgement of the jugular and other veins. After a few ounces or a pint of blood has been removed the lividity will pass off, the breathing become tranquil, and often refreshing sleep ensue.

One of the most distressing symptoms of heart disease is the sleeplessness already mentioned. For this opiates may be given, not only with safety but with the greatest benefit. One sixth or one fourth of a grain of morphia may be taken at bedtime, or if it produce gastric disturbance, it may be injected hypodermically. When all remedies have failed to act and the venous obstruction is so great that the whole body has become dropsical, the only resource remaining is to let the fluid run away as it collects, and this drainage may sometimes prolong life for many months. The usual method is to puncture the legs and so allow the fluid to escape. Sometimes the skin ruptures spontaneously, or vesicles form and burst until large sores or ulcers are produced, which continually discharge and keep up the necessary drain. There is always fear under these circumstances, whether the skin be broken spontaneously or artificially, of an erysipelatous inflammation being set up and quickly bringing about the fatal end.

Just as digitalis is the true remedy in the weak, irregularly acting heart in order to steady and strengthen it, so is it almost valueless when the organ is hypertrophied and acting steadily. It is therefore of no value in uncomplicated aortic disease with its accompanying hypertrophy. When, however, the enlargement has advanced to that extreme degree that the left ventricle can no longer contract, and therefore the mitral valve is unable to close, all the conditions of primary mitral disease exist. The action of the heart may then become irregular and venous engorgement with dropsy ensue. Then digitalis may be usefully administered; whenever, in fact, in aortic disease the pulse becomes feeble, frequent, fluttering, and irregular, as in mitral cases.

In the earlier stages of aortic disease, when the heart is steady, very few remedies are of use, although tonics are sometimes beneficial, as ammonia and senega or iron. Occasionally, when the blood-vessels are much relaxed, belladonna has been found useful. If in aortic cases, symptoms of angina pectoris occur, then the same remedies may be given as are found useful in that disease, such as the inhalation of nitrite of amyl or the internal use of nitro-glycerine (p. 16).

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* [Convallaria majalis has been recently revived as a remedy in similar conditions to those in which digitalis is valuable, and is undoubtedly efficacious in raising blood-pressure and thus producing diuresis.—Ed.]

DISEASES OF THE BLOOD-VESSELS

ANEURYSM.*—*Definition of the term—Varieties—Anatomy—Origin—Symptoms—of aneurysm of the ascending aorta—of the arch—of the descending aorta—Treatment—Abdominal aneurysms—Origin—Diagnosis—Treatment—Dissecting aneurysms—Cardiac aneurysm.*

INTRATHORACIC TUMOURS.—*Of the mediastinum—of the lung.*

THROMBOSIS AND EMBOLISM.—*Causes of coagulation—Effects—Pulmonary embolism—Fibrinous infarction—Cases.*

EXOPHTHALMIC GOITRE. —*History—Ætiology — Symptoms — Pathology—Event—Treatment.*

The physical conditions under which the thoracic organs are placed, render the clinical detection of tumours within the chest a more difficult and a less certain matter than it is in either of the other great cavities of the body. For the walls of the thorax being formed by a bony cage, one can seldom feel a tumour through them, as one can through the yielding abdominal parietes. And the contents of the thorax being largely constituted of the soft and yielding lungs, the effects of pressure are not, as in the interior of the cranium, almost necessarily perceptible, even while a tumour is still small, and they may be absent long after it has attained a very considerable size. These reasons further explain the fact that it is sometimes impossible to carry the diagnosis of a case beyond that of "intrathoracic tumour," using this term in its widest sense, to include aneurysms and some other affections, as well as new growths: and I think that they justify me in taking all these various kinds of disease together in the present chapter, although pathologically some of them are wide apart.

INTRATHORACIC ANEURYSMS.—An aneurysm is a circumscribed tumou containing fluid or solid blood, communicating directly with the canal of an artery and limited by the membrane called the sac. There has long existed a question amongst authors as to the need of any further qualification of this definition: whether it should be made to include the case where the whole calibre of the vessel is dilated, and whether in the case of the more circumscribed form it should have any reference to the number of coats involved. As regards the former part of the question, the terms *aneurysm* and *dilatation* are used as expressions of degree; a mere bulging of one side of the aorta would be spoken of as a dilatation, whereas a general dilatation of the whole circumference of the vessel if limited in length would be called a *fusiform* aneurysm. Not infrequently one side of the aorta may be so much enlarged as to produce symptoms by pressure on neighbouring parts, and then the expression "aneurysmal dilatation" is often used. This term is one of a practical and clinical significance, as it would not only include a case where the symptoms were referable to the impeded circulation due to dilatation, but also one where they showed the mechanical pressure of a circumscribed aneurysm. More commonly when we speak of "aneurysm" we imply the existence of a circumscribed swelling of the *sacculated* kind, communicating with the vessel by a distinct aperture.

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* [The first portion of this chapter, down to p. 77, which was left unfinished by the author, has been kindly supplied by Dr Wilks—and also the section on Thrombosis and Embolism, pp. 84–6.—ED.]

It has been a custom to divide this form of aneurysm into two kinds, *true* and *false*, according to the number and nature of the coats. It is a useless division, both because the terms have been used in opposite senses and also because the same aneurysm may in the course of time vary with respect to the number of these coats. Scarpa, one of the earliest and most original writers, found, what is correct, that in most aneurysms some of the coats were wanting, and thus he distinguished them from mere dilatations or false aneurysms. Subsequent writers, apparently guided by the ætiological sense, used the terms true and false as indicative of the perfection or absence of some of the coats of the vessel. It were better therefore to avoid these terms in any sense of this kind and to use the word false (if adopted at all) as synonymous with *diffused*, that is, to denote the case where the blood has burst through the real coats and becoming effused in the tissues around has formed out of them new walls wherewith to circumscribe the blood.

Probably most aneurysms arise from a morbid softening of the inner coat whereby a bulging occurs which pushes the middle and outer coat before it. When the aneurysm has reached any size it is found that the inner coat has become much attenuated or in parts altogether atrophied. The internal coat may be sometimes seen over the whole of the inner surface, and continuous with that of the blood-vessel; in other cases it ceases abruptly at the neck of the sac, or only patches and shreds of it are discoverable over other parts of the interior. Very frequently we find the whole inner coat smooth, but it differs from the original endothelium, and is bound up intimately with the coats below. Whilst the internal and middle tunics are thinned or destroyed, the outer one becomes much thickened, and in many cases constitutes the real sac of the aneurysm. It were in vain therefore to limit the term aneurysm according to the number of coats of the sac or to their perfection or deficiency. In the fusiform aneurysm with smooth walls there is no tendency for the blood to coagulate, but in the circumscribed form with the narrow orifice and roughened interior, coagulation is ever ready to occur. This is also promoted by the greater sluggishness of the circulation within it. When these more circumscribed aneurysms are examined, the interior of the sac is seen to be filled wholly or partially with fibrin: this is arranged in concentric layers, the outer, thin, hard, and pale, and interwoven with the coats, whilst the inner are thicker, softer, and darker. This shows that the deposition has taken place from the coagulation of blood and not from any exudation from vessels or from the walls of the sac. Nor does there seem to be any vascular connection between the sac and the fibrous clot. The mode in which this lamination of fibrin occurs is not very clearly ascertained; in the cases of very rapid cure by pressure the sac is merely filled with a uniform coagulum; and therefore the filling of the sac need not necessarily occur layer by layer, although out of this coagulum distinct lamination appears subsequently to take place.

Ætiology.—Aneurysms arise from special and definite causes, and are not due to the general changes found in the vessels from senile and other degenerations, for they are met with in the young, in the middle-aged, and in those who are otherwise healthy. In the smaller arteries aneurysms have been demonstrated to arise in connection with embolism.* Generally, however, there is some inflammation or atheromatous change in the inner coat, and it is probable that this may arise under the influence of gout, rheumatism, or alcohol. It has for some time been conjectured that syphilis also may be a cause, and there can now be no doubt from careful observation in all countries that this is by no means an infrequent antecedent. Then again a strain or violent exertion may seem to have been the precursor of aneurysm, but whether by producing an actual rupture of the coats or by setting up an inflammatory process is not very evident. It is a well known fact that the thoracic aorta is very liable to be affected in those who take violent exercise, especially in those who use their arms in rowing. In such persons the vessels become much altered in configuration, the coats thickened, and the interior atheromatous; thus aneurysmal sacs are liable to occur. The aortic valves also are apt to be affected under the same conditions. These are the causes which are supposed to be especially in operation in soldiers, who are found to be very liable to aneurysm; but it must be remembered that, besides the drill and other exercises, syphilis may constitute a very important share in their production. That violent exertion is productive of aneurysm is shown in the greater liability of men than women to the disease. Abdominal aneurysm, for example, is very rare in the female sex. Probably lead poisoning may be an occasional cause, for it is remarkable that the workers in this metal not only suffer from gout but from the diseases which so often accompany gout, as granular kidney and diseased blood-vessels; consequently they have been found to suffer not infrequently from aneurysms.

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* [In a case under my care in 1883, ulcerative endocarditis was seen to produce first embolism and then aneurysms of the radial and posterior tibial arteries successively.—Ed.]

Symptoms.—These depend for the most part upon the position of the aneurysm and the consequent disturbance of the neighbouring organs which are implicated. It is therefore natural to treat of the thoracic and abdominal aneurysms separately and then to further divide the former into those of the ascending part, of the arch, and of the descending thoracic aorta.

Aneurysms of the *ascending aorta* are strikingly different from those of the arch on account of the enlargement tending towards the front of the chest and therefore not implicating the important structures which aneurysm of the other portion of aorta necessarily do. If the aneurysm form a pouch in the sinuses of Valsalva the valves become involved, their closure may be prevented, a diastolic bruit be produced, and all the other signs and symptoms dependent upon regurgitation occur. These may indeed be the only symptoms, and none exist indicative of the presence of an aneurysm. The sac never grows to any great size and usually bursts into the pericardium and causes instant death.

When the aneurysm is somewhat higher up, it still may interfere with the efficiency of the valves and thus produce the before named symptoms, but if not, it will continue to increase, and often without much inconvenience, until its presence is known by a swelling or projection of the chest. Very often previous to its appearance pains in the chest might have been experienced, but not necessarily of a severe character. When it has come forward it may be recognised as a swelling or bulging of the chest on the right side over the second or third cartilage. When the hand is placed over it a distinct pulsation is felt, this is synchronous with the heart's action, so that when the other hand is placed over the apex two synchronous pulsations are found in the two parts of the chest. Sometimes the hand detects a thrill or *frémissement.* If the swelling should project so far forwards as to be encompassed by the hand a distinct expansion can be experienced. This expansile nature of the swelling is the most important and distinctive feature of an aneurysm. Without it there is little certainty of its nature, for a pulsating tumour may be nothing more than a growth situated over an artery, and the bruit which is present produced by pressure on the vessel; but no dilatation felt under the hand which grasps it would occur. The expansion may sometimes be well shown by covering the swelling with a piece of plaster in which a slit has been cut down its centre, if this be narrowly watched the slit will be seen to widen at every beat of the sac. If in the early stages a pulsation is felt, but not seen by looking directly at the chest; it may often be clearly observed by placing the eye on a plane with the patient's chest, either by looking over his shoulder when he is in the erect posture or by stooping the head to a level with his body when he is supine. On applying the ear a distinct throb or jar is communicated to the ear, and sometimes a murmur is heard. This is, however, by no means always the case, as it depends upon the relation of the sac to the vessel, and according to recent explanations of the causes of bruits would be due to the existence of a constricted opening through which the blood passed into a larger space beyond. It is very rare to hear a diastolic bruit, and if it exists must depend upon some peculiar and exceptional circumstance. As a rule the second sound is not only clear, but accentuated owing to the increased tension of the valves. As regards the heart, it is not as a rule enlarged in aneurysms of the aorta; it is only when an aneurysm exists at the very commencement of the aorta near the valves that any enlargement occurs, and then the apex beat would be found somewhat lower than natural. Occasionally these aneurysms press upon the vena cava and then some enlargement of the veins of the neck may be observed, or the surface of the chest may be seen covered with distended veins. They may also press upon the bronchus and impede the entrance of air into the lung. Sibilus and rhonchus would thus be produced, and if ulceration took place into the tube, hæmoptysis. If the aneurysm approach the axilla, the subclavian vein may be involved, and the nerves of the brachial plexus, so as to cause pain and swelling of the arm. Sometimes aneurysms of the ascending aorta push themselves forward and absorbing the ribs or passing through the sternum project on the outside of the chest as circumscribed tumours; sometimes they are of a very large size; at other times rounded like an orange. The skin may become thin and red but the aneurysm very rarely bursts externally. The percussion note over an aneurysm is of course dull, the extent varying with its size.

In aneurysm *of the arch of the aorta* the symptoms are more numerous and more important than those of the ascending part. This is owing to the fact of many other structures being implicated. An aneurysm at this part produces all the mechanical effects of a tumour, and this is the reason why new growths and aneurysms are so constantly mistaken for one another, for if the latter be deep seated the characters peculiar to them may be altogether wanting—that is pulsation combined with a bruit. If we consider the close packing together of the aorta, trachea, bronchi, œsophagus, pneumogastric, recurrent laryngeal and sympathetic nerves, it is evident that any tumour like an aneurysm forming amongst them must interfere with these structures. If it touch the spine, the bones may be eaten away. The pulmonary artery may also be pressed upon or even opened. The parts usually involved, however, are the trachea or bronchus. These being for some time pressed upon, subsequently ulcerate until the mucous membrane is reached and an oozing of blood takes place. If the aneurysm contain much fibrin and the solid portion be in contact with the air-passages, this oozing of blood or occasional hæmoptysis may continue for a long time, even for weeks or months, and in some exceptional cases for years. Sooner or later, however, the blood breaks through in larger quantities and thus causes immediate death.

If the aneurysm form in the arch behind the sternum, it soon shows itself on the left side as a pulsating tumour about the second left cartilage. If there have been no symptoms, this when first observed may be mistaken for the auricle. As in the case of the aneurysm of the ascending aorta, it may be seen and felt to pulsate and occasionally a bruit is heard. Should the aneurysm remain behind the sternum or be formed beneath the arch no external signs of it are apparent. Then all the symptoms are due to its pressure on the parts which surround it. One of the most common is difficult breathing, this may arise either from pressure of the trachea or bronchus or from implication of the pneumogastric nerves. In the former case there is, as before said, evidence of the pressure by sibilus, and by mucous expectoration tinged with blood. In other cases the difficulty of breathing is due to paralysis of the larynx or its vocal cords. It is usually the left nerve which is involved, which may be found after death closely incorporated with the coats of the sac, and perhaps much thinned; under these circumstances the muscles of the larynx, especially the posterior crico-arytenoid, are much atrophied. The symptoms due to pressure on the nerve generally differ from those which are associated with direct pressure on the air-passages, the difficulty of breathing being paroxysmal and the cough peculiar. It is like that of a croupy child, being ringing or brassy, and in the intervals the breathing may be free and natural. If the laryngoscope be used it will be found that one of the vocal cords, generally the left, is paralysed and motionless; and sometimes, although only one nerve is involved, both cords become paralysed, causing great difficulty of breathing and threatening suffocation. It must be remembered that during the act of breathing the larynx is actively opened, and therefore if the muscles be paralysed it would close during inspiration.*

Pressure on the œsophagus may cause difficulty of swallowing, and pressure on the pneumogastric and pulmonary plexus may set up a low form of pneumonia. It is frequently found after the existence of sibilus, and pressure on the lung, that febrile symptoms ensue, and that one lung is becoming solidified; this slowly brings the patient to an end. The lung is then found to be hepatised or in a state of purulent infiltration, and the cause of this is no doubt to be explained, as Sir William Gull and Dr Budd long ago observed, by an implication of the nutritive nerves.

The implication of the sympathetic nerve in the neck may affect the pupil of the eye, its paralysis causing contraction of the pupil in the same way as paralysis of the third nerve causes dilatation. The pupils ought, therefore, to be carefully examined in cases of suspected aneurysm.

As regards pain, this varies according to circumstances. As a rule, it is a pain in the chest or down the arms which first takes the patient to the medical man. This in course of time may become very acute and accompanied sometimes by an actual weakness of the arm. The pain may have its origin in the aorta and so, through the sympathetic and spinal nerves, is at last referred to various parts of the chest and upper extremities. If the aneurysm be situated at the lower part of the arch it may erode the spine and so immediately involve the nerves passing to and from it.

Another important indication of the existence of an aneurysm may sometimes be found in the pulse; and especially in one pulse being smaller than the other. This may arise from three causes, the subclavian artery may run outside the sac and be compressed by it, or it may come off from the sac and its mouth be closed by fibrin, or, more frequently, the mouth of the artery is open but being given off from the aneurysm, the latter, being deficient in the normal tension of the aorta, fails to produce the natural pulsation in the vessels proceeding off from it. It must be remembered that when the blood is delivered by the ventricle to the aorta the latter contracts upon the blood and every shock is felt in the distant arteries, but if the aorta were converted into one large sac the impulse of the heart would be lost in this space, and the blood would flow out through the efferent vessels in a continuous stream. The apparatus would resemble that of a spray instrument where the intermittent action of the first bag is followed by a continuous action of the second. Lately a case existed in Guy's Hospital which exemplified this : the pulses at the wrist became quite imperceptible but the warmth of the hands and their vascularity showed that the blood flowed into them as before. It is the exception, however, to find, as in this instance, the pulse quite cease, the more usual occurrence is for that in one wrist to become more feeble than that in the other. If the aneurysm should involve the innominate the right pulse would be enfeebled, as would be the left if the aneurysm should be in front of the left subclavian. The sphygmograph is found to be very useful in demonstrating on paper the difference between the perfect pulse of one wrist with all the parts of the natural tracing and the imperfect delineation of the other.

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* [On this subject see, however, Dr Bristowe's remarks in the 3rd vol. of the 'St Thomas's Hosp. Reports' (1872).—Ed.]

Aneurysms of the *descending thoracic aorta* are necessarily productive of different symptoms from those of the arch. They soon involve the bones of the spine, which they erode and expose the nerves which proceed from it. Pain, therefore, is one of the commonest symptoms and may be the earliest. It is often very defined, as in the course of a particular intercostal nerve, and so the seat of the disease may be accurately determined. The thoracic aneurysm may also involve the lung, or press upon a bronchus, or at other times may compress the œsophagus. If it advance, it may make its way backwards, and involving the ribs be felt at the back ; it may even enter the canal and compressing the cord produce paraplegia (vol. i, p. 412). Under these circumstances the case is clear, the pulsating tumour may be felt and seen, and a bruit be heard with the stethoscope. Death may occur from implication of the lung causing pneumonia, or by rupture of the sac into the œsophagus, but more usually by its bursting into the pleural cavity.

Treatment.—The object which the physician has in treating aneurysms of the aorta is the same as the surgeon has in view in treating aneurysms of a limb, to retard the circulation in the vessel and aid the deposition of fibrin. This is more likely to be effected when the opening of the sac is small and the interior roughened. In the case of a popliteal aneurysm the limb is bent or the femoral artery is compressed, and so the blood coagulates ; and the same object is attempted in the case of the aorta. The method of cure is therefore to lessen the rapidity and force of the circulation, and, if possible, to increase the deposit of fibrin from the blood. The patient must be kept absolutely at rest in the recumbent posture, and be subjected to a strict diet. The original method proposed by Valsalva, was bleeding the patient several times and keeping him on the smallest amount of food. This plan is deprecated by Tufnell, who, however, has revived the method in a modified form with remarkable success. The object he allows is to reduce the circulation and keep the blood in a highly fibrinised state, but this is not accomplished by starvation. His experience has been mainly with abdominal and local aneurysm, but the same method equally applies to aneurysms of the thoracic aorta. Tufnell allows three meals a day and a small amount of fluid with absolute rest for at least two months. The value of rest in the recumbent posture he illustrates by the fact, that in one patient the difference of the number of beats of the heart between the sitting and recumbent position was thirty-five per minute. This, he says, is equal to more than 50,000 in the twenty-four hours, and no remedy in the Pharmacopœia could produce so striking an effect. The diet he recommends is the following: for breakfast, two ounces of bread-and-butter and two ounces of milk or tea ; for dinner, three ounces of mutton and three ounces of potatoes or bread, and four ounces of claret; for supper, two ounces of bread-and-butter and two ounces of tea: the total per diem being ten ounces of solid food and eight ounces of fluid.

Having considered the more important question of rest and diet, we come to another as to the value of medicine. Digitalis is often given, but without any striking effect, so also lead, ergotine, and other remedies; the only one which can be spoken of as having any real efficacy is the iodide of potassium. This is now given in large doses, as ten or fifteen grains, three or four times a day for several weeks and sometimes with marked success. Under its use the aneurysmal sac is found to become hard, as if the remedy favoured the deposition of fibrin. Whilst this is taking place the symptoms, such as pain, are subsiding, which show that the pulsating sac produces symptoms which are not due to the tumour alone, for when the latter has become hard and inert all the troubles disappear. It is true that the cured aneurysm is smaller than the active one, but size alone will not account for the subsidence of symptoms. The fact is well seen in cases of popliteal aneurysm where the pain and tenderness immediately cease after the arrest of the circulation through the sac.

Various other local measures have been attempted but not with much success, as, for example, galvanism. The poles of the battery have been passed into the sac so as to produce coagulation of the blood. Ligature of the distal vessels has been several times performed and in some cases with temporary benefit. In cases of aneurysm of the aorta ligatures have been placed on the left carotid or on the carotid and the subclavian conjointly.

Abdominal aneurysm.—Aneurysms of the abdominal aorta occur more frequently at some parts of the vessel than at others. The most common site is just below the diaphragm at the origin of the cœliac axis; they are more rare at the origin of the superior mesenteric artery and are very seldom found lower than this point. The first-mentioned position is the most common, and the aneurysm here often involves the cœliac axis. This variety is said by Sibson to constitute 70 per cent. of all abdominal cases.

The first symptoms of abdominal aneurysm are generally painful feelings in various parts of the abdomen. These may be lancinating, paroxysmal, or encircling the body. They may be due to the sympathetic nerve being involved in the sac or to the connection of these nerves with the spinal, or the latter may be directly involved by the aneurysm eroding the vertebræ. If the patient have an aneurysm in the upper part of the abdomen it would be most usually found in the left hypochondrium, or if nearer the median line, more to its left side. A distinct tumour may be felt expanding under the hand or a mere pulsation; it may be so great as to give considerable movement to the stethoscope when placed upon it. If not only an impulse but an expansion be also felt the diagnosis of aneurysm is at once made. Sometimes also a thrill can be felt. On auscultation a systolic bruit may also be heard. If the aneurysmal tumour be below this, it can still more easily be grasped and its nature made out. If it grow backwards it may also be detected in the loins both by its pulsation and the existence of a bruit. Various other symptoms may be present owing to its pressure. Thus occasionally actual obstruction of the intestine has occurred; constipation, however, is so frequently observed, that this probably arises not from direct pressure but from paralysis of the sympathetic nerve. A very careful examination of the crural arteries will sometimes show a slight retardation of their beat as well as a diminished tension. The mode of death in abdominal aneurysm is generally by rupture; this may occur directly into the abdomen causing instant death, or it may occur behind the peritoneum, whereby the blood becomes effused into the areolar tissue and a coagulum is temporarily formed. This new or false sac soon, however, gives way with the same result as in the first instance. Very rarely and exceptionally the coagulum becomes fibrinous, and this being incorporated with the original aneurysmal sac, a cure is effected. Other exceptional terminations of abdominal aneurysm are sometimes met with, as in a patient lately in the hospital in whom a loud whizzing bruit was heard in the lower part of the back, and who also had dropsy of the lower extremities. After death a small aneurysm was found communicating with the vena cava constituting an aneurysmal varix. The venous blood was retarded in its flow upwards and so the dropsy was produced.

Ætiology.—The causes of abdominal aneurysm are the same as in the thoracic form. Violent exertion is no doubt a very common cause, and this is the reason why it is so more common in men than in women; for in women it is exceedingly rare. In several cases there has been well-marked syphilis, and no doubt can exist as to this being a frequent cause. Of three female cases in Guy's hospital two had had syphilis; one of them occurred in 1853 under the late Dr Babington. She was a prostitute and was being treated for syphilis when she died suddenly from rupture of an abdominal aneurysm at the cœliac axis.

Diagnosis.—This is not always easy, since pulsating tumours may exist in the abdomen which are not aneurysmal, and bruits may be discovered in the aorta which have not this origin. In women especially who are anæmic frequent pulsations in the abdomen occur, and bruits are heard, which suggest aneurysms. Then, again, a tumour may be raised up by the aorta and pulsate, and in it a bruit may be heard, but these two signs are not sufficient to warrant the diagnosis of an aneurysm; nothing but the expansile nature of the sac can afford a distinctive diagnosis. If the patient be placed on his hands and knees and the tumour be handled, any falling forwards on its detachment from the back would be in favour of a growth, whilst an aneurysmal sac would still remain in its place.

Treatment.—The general treatment of abdominal aneurysms is the same as that of thoracic, restricted diet, absolute rest, and drugs. Besides these methods the opportunity occurs in abdominal aneurysms of using local means, as mechanical pressure. The method has been most successfully carried out by Dr Murray, of Newcastle (1863), and followed by others. He found that sometimes a very short period was sufficient to cause coagulation in the sac and the cure was most rapid. The aneurysm must be sufficiently low down so as to enable the surgeon to apply pressure above it. Unfortunately, the more common form at the cœliac axis is too high to allow room of any instrument being applied above it. In Dr Murray's first case ('Med.-Chir. Trans.,' vol. xlvii, p. 187) pressure was used for two hours, and again for five. The aneurysm was rapidly cured, and the man remained well for six years afterwards. It was found that the aneurysm came off from the aorta at the origin of the inferior mesenteric artery; this was obliterated and the circulation had been carried on through collateral branches. This case was followed by one of Dr Greenhow's, in which also one application was not sufficient; pressure was continually repeated for a week and at last a perfect cure resulted. Mr Durham reported a case about the same time of an abdominal aneurysm cured by pressure. This was kept up with the patient under chloroform for ten hours until pulsation ceased. The circulation then also stopped in the femoral arteries and coldness of the legs came on. Pulsation again occurred in the aneurysm but in a less degree. The treatment was continued but pulsation was not completely arrested in it for a month. Perfect recovery soon followed. Another case occurred in Guy's Hospital where pressure was kept up for some hours on the distal side of a high aneurysm. No apparent effect upon it was produced but collapse came on and in twenty-four hours the patient died of peritonitis. The intestine was found bruised and covered with lymph. The sac contained a coagulum which appeared as if it had been deposited during life.

Dissecting aneurysm.—In connection with aneurysm it is necessary to allude to the subject of rupture of the aorta owing to disease of its coats. It is rare for the whole of the coats to give way simultaneously, it is more usual for the inner coat to lacerate and the blood then to find its way between the coats, producing a dissecting aneurysm, and finally for the outer coat to rupture causing the patient's death. When the blood is effused in this manner it tears asunder the layers of the middle coat so that a part is found united with the intima, and a part externally covered by the adventitia. Two cases may be mentioned in illustration of this remarkable form of lesion.

A former sister of Petersham Ward, about sixty years of age, was seized a month before her death with a violent pain across the chest and abdomen, her heart became quick and tumultuous, and it was thought at the time that she was dying. She, however, rallied, had some slight febrile symptoms, and in a few days was doing duty in her ward. Two days before her death she had another similar attack and again quickly recovered. On the day of her death she fell dead whilst dressing. The post-mortem showed the pericardium to be full of blood, which had proceeded from a rent at the commencement of the aorta. On the inside the rent was seen to be an inch long just above the valves, and through this blood had passed between the coats of the vessel; outside was the rupture of the external coat, but this did not correspond to the internal. Some distance above this and at the beginning of the arch was another fissure, an inch long and older in date; it was also transverse, had smooth edges connected by bands, and had quite healed. Proceeding from this was effused fibrin in the coats of the artery, which were separated from one another to the end of the abdominal aorta. The separation had occurred in the whole circumference of the vessel.

Another example was that of a gentleman about sixty years of age. He was a merchant, and after returning from the city and eating his dinner was seized with a severe pain in the chest. It continued all the evening until he went to bed. He was found afterwards dead at the side of his bed. The autopsy showed the pericardium full of blood which proceeded from a fissure in the aorta. On examining the latter from within a laceration of the vessel was seen to have occurred half an inch above the valves, it was transverse and ran almost completely round the vessel. Blood had passed through and separated the coats throughout the arch, the thoracic and the abdominal aorta. It had, however, taken only about two thirds of the circumference. The clot was quite recent. The laceration in the outer coat was at right angles to the internal one and was an inch and a half long.

Dr Peacock, who thoroughly investigated the subject of dissecting aneurysm, speaks of a variety occasionally met with of a chronic nature, in which a long time supervenes between the rupture of the internal and external coats, so that a distinct pouch may form in the external coat of an ordinary aneurysmal character. This becomes in time lined by a smooth membrane.

Aneurysms of the heart.—Although these are not capable of being diagnosed during life, and therefore the subjects of them cannot be of any interest from a clinical point of view, it is important to have a knowledge of their existence. Already, under the heading fibroid degeneration, dilatations of the ventricles have been spoken of (p. 36), and some of these being sacculated may be styled aneurysms. As a fact, all the cavities of the heart may present distinct dilatations or aneurysmal pouches; they are exceedingly rare, however, elsewhere than in the left ventricle. Here they are met with in all parts, on the sides and at the apex. They may be mere pouches in the wall of the ventricle without any projection externally, or they may be seen as distinct sacs on the surface. In the former case it is a question whether they have arisen as acute aneurysms or ulcers. They are always associated with a fibrous change in the surrounding muscular tissue, so that it seems evident that they have their origin in a softening or degeneration of fibrous tissue the result of a myocarditis. The inflammation of the pericardium, however, on the exterior by no means proves the existence of a prior pericarditis, as this might arise subsequent to the formation of the aneurysm. Sometimes the aneurysmal sac has become "cartilaginous" or calcified, and thus undergoes a natural cure. As some fibroid deposits are syphilitic, aneurysmal cavities may have had their origin in this tissue.

INTRATHORACIC NEW GROWTHS.—From a clinical point of view, the new growths that occur in the thoracic cavity have to be divided into two main groups: (1) some of them are seated in the mediastinal tissues, or at least implicate the root of the lung on one or on both sides; (2) others affect only the substance of the lungs, perhaps reaching the pleural surface, but not interfering with their roots.

Mediastinal new growths.—The tumours that I am about to describe differ in different cases in almost every particular: in their starting-point from one or another of the thoracic structures, in their histology, and in the various directions in which they may grow. It may therefore seem illogical to group them together under a single heading; but there is really no alternative, because no clinical distinction can anywhere be drawn between them. As regards the origin of a mediastinal growth, indeed, it is seldom possible to come to any positive conclusion at the time of an autopsy, when it has generally reached a great size and involved various tissues. The common supposition is that they very often start from the bronchial and other lymph glands; and this view is doubtless correct for many of those cases in which the thoracic affection is only part of Hodgkin's disease or general lymphosarcoma, as well as for those in which it is secondary to a primary growth situated in some other part of the body. But I have on more than one occasion noticed that the glands have been only partially attacked: some have been infiltrated with the new growth, but others, though imbedded in it, have retained their natural structure. In such cases it is natural to look for some other seat of origin, and this may be assumed to be either the thymus or the mediastinal connective tissue or fat, or perhaps the pericardium, or (sometimes) the periosteum of the sternum. As for the histology of mediastinal tumours, the vast majority of them are made up principally or entirely of small round cells, and are classified by some observers as "lymphomata," by others as "round-cell sarcomata." But some specimens contain a large proportion of spindle-cell tissue, and others consist of little but fibrous tissue, and some have been described as having an alveolar structure, and even as being true carcinomata. In yet other cases, mediastinal tumours have been found to be of a syphilitic nature, being formed of gummata imbedded in a dense fibrous material. And lastly, some have been "dermoid cysts," containing hair and bone, and even teeth, besides a quantity of fat.

Among the relations which mediastinal new growths bear to the various structures contained in the thoracic cavity, by far the most important are those that concern, on the one hand, the great systemic veins, and, on the other, the main air-passages. Indeed, I think that it is almost a peculiarity of the affection now under consideration to produce obstruction either of the *vena cava superior*, or of one or other of the innominate veins, or of all three of them. I have notes of one case in which a tumour in the lower part of the thorax reduced the orifice of the *vena cava inferior* to a mere slit, through which the finger could scarcely be passed. As I have already observed at p. 71, aortic aneurysms occasionally, but comparatively seldom, interfere with the veins; and almost the only other cases in which the flow of blood through the *vena cava superior* is retarded or prevented are certain cases of heart disease in which, during an acute attack of pericarditis, there has been inflammation of the mediastinum with thrombosis of one or both of the innominate veins, ending in obliteration of the affected vessels. But when there is a mediastinal new growth venous obstruction is a very frequent result. Sometimes the growth penetrates the coats of the vena cava, and fungates within it as a soft smooth mass which may be as large as a thumb. Sometimes it surrounds that vessel or one of the innominate veins, and causes extreme narrowing or even complete obliteration of the blood-channel. In either case there may be a consecutive thrombosis of the jugular and other tributary veins. The clinical effects of these lesions is sometimes very marked. There may be great œdema of the arm and hand on one side or on both. The neck and the face may be enormously swollen and of a deep red or purple colour, with obvious over-distension of their veins. When the obstruction is limited to the superior cava, a collateral channel for the passage of the blood may be afforded by the azygos vein which becomes dilated. But when both innominate veins are blocked, the intrathoracic vessels can do little towards carrying on the circulation, which then has to depend in great measure upon anastomoses between the superficial veins of the chest and back with those of the lower part of the trunk. The consequence is that the body becomes covered with dilated vessels, and may acquire a deep purple or claret colour. One can generally easily make out in which direction the blood has to flow, for when one of the veins is emptied by pressure along its course it fills far more rapidly from above downwards than from below upwards. Or passing a piece of string round the chest, one may see at once that the vessels above it remain full, whereas those below it become empty. But in many cases the appearance of the affected parts is in itself a sufficient indication. For it is a curious fact, doubtless dependent on the presence of valves in their interior, that obstructed veins are apt to become far more tortuous when the circulation through them is in the reverse, than when it is in the natural, direction. Thus when the superior cava is blocked the veins may be zigzagged and varicose all over the chest and the upper part of the back, whereas those over the lower part of the body may be a little larger than natural, but almost straight. If there is obliteration of the inferior cava, on the other hand, the effect may be exactly the converse. Watson, in his sixty-third lecture, records and illustrates by diagrams two cases in which this distinction was very manifest. The obliteration of the superior cava in the first of these cases was, however, due to an aneurysm, that of the inferior cava in the second case to an hydatid cyst of the liver. Among the subjective symptoms noticed by the former patient were nervousness, distressing dreams, and giddiness on stooping, which also made his face and ears turn black. A man who died in Guy's Hospital of a mediastinal new growth in 1868, said that the first indication that anything was wrong with him was that a sensation of swimming in the head came on when he stooped.

In speaking of stenosis of the lower air-passages in general, I have already discussed (vol. i, p. 829), the effects of compression of the trachea or of the bronchi by mediastinal new growths. The records of post-mortem examinations at Guy's Hospital seem to contain very few cases in which these parts are stated to have been found entirely untouched by the disease at the time of death. But sometimes it is noted that although their walls, even to the mucous membrane, were completely infiltrated, there was yet no narrowing of their calibre. In most cases the growth extends along the bronchi into the pulmonary tissue to a greater or less extent. Sometimes it fills up a very large part of the lung, forming large masses in its interior, and even reaching its surface and thickening the pleura or adhering to the inside of the ribs. For obvious reasons the disease is then either confined to one side, or at least far more marked on one side than on the other. Dr

Powell, in Reynolds' 'System,' says that the left lung is invaded more often than the right, but among twenty-six cases that I have taken from the post-mortem records of Guy's Hospital I find that the numbers on each side are nearly equal.

The *physical signs* caused by mediastinal new growths are due chiefly to their size, and to their interfering with the air-passages and with the lungs. Their bulk is sometimes enormous. I have notes of one case in which there was a solid mass that weighed ten pounds, and of another in which the measurements in three dimensions were ten inches, seven inches, and five inches respectively. Such great tumours naturally cause considerable enlargement of that side of the chest which they principally occupy. The intercostal spaces may be widened and flattened; the movements of the ribs may be much impaired. But, on the other hand, if the growth is attended with shrinking of the lung, the measurements may be less than those of the opposite side. In some cases the tumour protrudes above the clavicle, so as to be felt at the root of the neck; in others it bulges through one or more of the intercostal spaces. It may lead to the absorption of bone to some extent, but it never (so far as I know) appears as a rounded swelling, projecting far beyond the natural level of the ribs, such as is formed by some aneurysms. It may, however, pulsate more or less forcibly, and a systolic murmur may be transmitted through it from the heart or the aorta.

As may well be imagined, such large growths as those to which I have been referring cause great dulness on percussion. This is commonly very marked over the sternum, and for a greater or less distance on each side of it. But it may also extend over the whole of the front of the chest on one side up to the clavicle, or over the back to a variable distance round one scapula, or between both scapulæ, or even over the whole of one half of the chest from apex to base, including the axillary region. Not only is the percussion-sound absolutely toneless, but the sense of resistance to the finger may be extreme. The condition may therefore be exactly like that which would be produced by liquid effusion into the pleural cavity; and as a matter of fact such an error of diagnosis has been often committed, and repeated exploratory punctures have been made, which of course have led to no good result. The best way to avoid this blunder is to map out carefully the area of dulness in all directions, especially over the middle of the chest in front, where it may be found to extend beyond the bounds to which it would certainly be limited were it due to pleural fluid. But the difficulty is increased by the fact that when the pleura is reached by a new growth, effusion often takes place, and sometimes in large quantity. Hence the success of a paracentesis is no warrant for resting satisfied with the diagnosis that there was liquid in the serous cavity; that may be merely a complication of a far more grave disease. A circumstance which should generally excite suspicion is the presence of altered blood in the liquid, giving to it a dark brown tint. But in some instances in which there is a mediastinal tumour the pleural effusion is straw coloured, and exactly like that which is seen under other conditions.

On the other hand, there are cases, especially where the growth is limited to the roots of one or both of the lungs, in which percussion yields altogether negative results.

The other physical signs of mediastinal tumours are very uncertain. Tactile vibration is sometimes increased, but I think more often lessened, or even entirely abolished. It is, of course, only in the latter case that the disease is likely to be mistaken for pleural effusion. There may be more or less loud bronchial breathing, or only a very faint vesicular murmur may be audible with the stethoscope when the patient inspires, or there may even be absolute silence. The stridulous and other sounds produced by narrowing of the lower air-passages have been described in the first volume, p. 833. In many cases there are râles, of varying quality, not over the tumour itself, but over parts of the lung to which pass the branches of an obstructed bronchus. And to these may be added the signs of consolidation, when inflammation of the pulmonary tissue sets in.

In many cases mediastinal growths invade the pericardium, generally at the base of the heart; and they may then spread downwards to a greater or less extent, both along the parietal layer of the serous membrane and also in the walls of one or both of the auricles. The pericardial sac may either become closed by adhesions, or it may become distended with liquid effusion, which is often sanguineous, or it may be affected with inflammation, leading to the exudation of lymph or of lymph and pus. Of the great vessels, the aorta almost always seems to escape entirely, the venæ cavæ (at least the vena cava superior) are very often greatly narrowed, as has already been pointed out; the main divisions of the pulmonary artery and of the pulmonary veins are also in many instances pressed upon, so that their calibre is much reduced. At first sight it might appear probable that cases in which the pulmonary vessels are involved should be characterised by more intense dyspnœa than would otherwise be present. But in practice this is not observed, nor ought it perhaps to be expected, inasmuch as Lichtheim has shown by experiment that one fourth of the natural calibre of the pulmonary artery suffices to keep the lungs fully supplied with blood. Even when the heart is not invaded by a mediastinal growth, it is often much displaced; generally it is pushed downwards and to the left, but sometimes, when there is shrinking and contraction of the left lung, it is dragged up so as to be felt pulsating not far below the clavicle. Although the aorta itself resists the pressure of the growth, it may happen that some of its branches are more or less narrowed, causing the radial pulse to be weaker and smaller on one side than on the other.

The œsophagus does not seem to be often occluded by mediastinal growths; at least in the reports of post-mortem examinations at Guy's Hospital I find very few instances in which this is said to have been the case, or in which dysphagia is noted as having been among the symptoms. One patient is reported to have brought up his food almost directly after attempting to swallow it; at the autopsy the œsophagus was found pushed aside, but not invaded. In some rare instances the disease extends into the spinal canal, causing a "compression-paraplegia."

One very curious effect of a mediastinal growth which I had an opportunity of observing many years ago, must, I think, be attributed to pressure upon the vaso-motor nerves. The patient was a girl, aged twenty-six, who was originally admitted, in the summer of 1866, into one of the surgical wards under Mr Cooper Forster for a defective state of the circulation in her fingers, which were blue, cold, shrunken, and also very painful and tender. After a few weeks she was discharged, but she again came in under Dr Moxon in the following April, and died some months later. At the autopsy it was found that the growth infiltrated the fibrous tissue in front of the spine, and involved both the first dorsal nerve, and the sympathetic trunk. In that instance nothing abnormal was ever detected in the state of the pupils. But in other cases in which there is pressure upon the sympathetic trunk on one side at the root of the neck, the corresponding pupil is smaller than the opposite one, especially when but little light reaches the eyes, so that both pupils should normally be dilated. The explanation is that the dilating mechanism of the pupils is worked through fibres contained in the sympathetic, but the contracting mechanism through the third nerve. Consequently when the sympathetic is paralysed, the natural balance is lost and the contracting fibres get the upper hand. In three cases of mediastinal growth, Rossbach is said to have further observed that both pupils dilated regularly with each inspiration; in two of these cases, in which there was a swelling above the clavicle, pressure upon this tumour caused the pupils to become widely dilated, while the frequency of the pulse also underwent a temporary alteration, being retarded in one instance and quickened in the other.

The other symptoms of mediastinal growths vary widely in different cases. There is generally more or less dyspnœa from the time when the patient first notices that anything is amiss with him. Very often he is obliged to sit up, even at night; sometimes the only position in which he can sleep is leaning forwards, or even lying on his face. The breathing is generally accelerated, twenty-four to thirty in the minute. Pain is commonly an early symptom, and sometimes (though not usually) it is very severe. It may be referred either to the side, or to the shoulder, or to the middle of the chest in front. Sir Risdon Bennett speaks of it as sometimes sudden and transitory, and attendant on physical exertion.* Most patients have a troublesome cough, which is often described as "ineffectual," giving rise to little or no expectoration. But sometimes a viscid mucus is brought up, and sometimes this contains blood, so intimately mixed with it as to give it an appearance like that of red-currant jelly, to which writers attach importance as indicating the nature of the disease. The spitting of pure blood, too, is not uncommon, and it may occur at the very commencement of the clinical history of the case, and more than a year before the fatal termination. Profuse hæmoptysis is seldom observed, but Dr Church has recorded in the 'Pathological Transactions' (vol. xix, p. 64) one instance in which four pints of blood were brought up immediately before death, though the bleeding probably came from broken-down lung tissue in the neighbourhood of the growth, rather than from the growth itself.

Cachexia is by no means a prominent symptom. The patient often looks well and ruddy for some time after he comes under treatment, and even at the last there is not often extreme emaciation. Pyrexia is generally absent, but Risdon Bennett relates a case (loc. cit., p. 121) in which the temperature had varied from 100° to 101·4°, but in which Dr Sutton, who made the autopsy, could detect no appreciable inflammatory changes, so that the only way of accounting for the febrile disturbance was by referring it to the active cell growth that had been going on, not only in the lungs, but also in other organs of the body.

To complete my account of the clinical effects of mediastinal tumours I may note that in two cases of dermoid cysts the patient has expectorated hairs, in one of them for as long as twelve years before death.

But sometimes, when there is a mediastinal growth, no chest symptoms are present at any period of the case, which is cut short by disease of some other part of the body, most frequently by a similar tumour of the brain. I have notes of several instances of this kind, and they are of interest, not only in themselves, but also as showing how little dependence can be placed upon the duration of chest symptoms up to the time of death, as an indication of the real rate of progress of the disease. On the other hand it must not be forgotten that even if we did theoretically know the time necessary for a growth to develop itself, from its very commencement until it destroys life, we should still be unable, so long as we cannot fix the date at which it begins, to make any use of the knowledge in clinical practice. In nine cases at Guy's Hospital in which the duration of the symptoms is noted, I find that it varied from two and a half to eleven months. The extremes in either direction are probably afforded by two cases cited by Hertz, in Ziemssen's 'Handbuch;' one of Grisolle's, which proved fatal in a week from the first appearance of symptoms; the other one of Eyer's, in which life was prolonged for at least seven, and possibly for fifteen years.

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* [Lumleian Lectures on "Intrathoracic Growths," p. 179. This refers rather to a sudden effort or injury producing a pain, to which the patient refers as his earliest symptom. The same author says, with reference to the later symptoms: "The amount of mere pain is seldom such as to call for the use of any large quantity of opium. But the distress is often very great" (p. 186).—Ed.]

With regard to the *causes* of mediastinal growths very little is known. In one or two recorded instances they have been attributed to injuries, such as blows upon the sternum; but it may well be doubted whether these were more than a coincidence. The most remarkable contribution to the ætiology of the disease that I know of is a paper by Hesse in the 'Archiv der Heilkunde' for 1878, where it is stated that in the mines of the Schneeberg 75 per cent. of all the miners—from twenty-one to twenty-four each year—die, generally about the age of forty, from "cancer of the lungs," spreading from the root. Professor Ernst Wagner examined some specimens of the disease and found that the growth is a lympho-sarcoma. Only two explanations appear possible; one that it is the result of a tendency inherited and transmitted from generation to generation; the other, that it depends upon the nature of the minerals worked in the mines, which contain bismuth, cobalt, and nickel, with some arsenic and sulphur. With regard to the first suggestion it is noteworthy that the miners of the Schneeberg are recruited from among the sons of former miners; whether intermarriages are frequent does not appear. Dr Walshe mentions the cases of two brothers who were each affected with intrathoracic growths, and I shall presently allude to what is perhaps a parallel instance. In favour of the second suggestion I do not know that any corroborative evidence can be adduced. But I have a vague impression that a good many of the cases that I have seen from time to time have occurred in engineers and in other artisans working with metals.

Most observers say that more men than women die of mediastinal growths, and this is confirmed by a collection of thirty-three cases that I have taken from the post-mortem records of Guy's Hospital; the proportion being more than two to one. As regards age, I find that the numbers for each decennial period from twenty to sixty are almost exactly the same; whereas, it is generally stated that the disease is more frequent in persons between twenty and thirty than in those who are older. A very few cases have been observed in children.

The *prognosis* in cases of mediastinal new growth is very grave. If recovery should take place in a case diagnosed as one of this disease, the general impression would be that a mistake had been made, and that the patient was really affected either with some inflammatory or fibroid or syphilitic thickening of the intrathoracic structures, or else with aortic aneurysm. But I have met with one instance which goes some way towards establishing a different conclusion. It is that of a man named John Bullions, who was admitted into Guy's Hospital under Dr Habershon on February 1st, 1867, with loss of voice, stridulous breathing, great swelling of the neck, œdema of the chest, and fulness of the veins. There was also slight deficiency of resonance on percussion over the right apex and over the root of the right lung behind. Under the administration of iodide of potassium he rapidly improved, and left the hospital on March 2nd, after which he returned to work. But on May 22nd he was readmitted with what was apparently an attack of erysipelas of the face and neck. This also quickly subsided, and from that time I lost sight of him, However, in 1871, another man, named Thomas Bullions, aged nineteen, came into the hospital and died of a mediastinal new growth, as was proved by the autopsy. Struck by his peculiar name, I inquired and found that the former patient was his elder brother and was then in good health, though still rather short of breath. Were both cases of the same nature, like those recorded in two brothers by Dr Walshe, or were they different?

In the *treatment* of this disease a faint hope of cure is by the administration of iodide of potassium, arsenic, or mercury. But when there is great venous obstruction, marked relief is often afforded by venæsection, cupping, or leeches. To ease pain, recourse may be had to blisters or mustard poultices, and also to the various anodynes; for the cough, Risdon Bennett recommends antimony in small doses with a sedative.

Pulmonary new growths.—Under the present heading I propose to place only those cases in which the lung is affected with a new growth that leaves its root free. Clinically they differ widely from the "mediastinal" cases that I have just been describing. Very often, indeed, they give rise to no symptoms at all. The position of the pulmonary in relation to the systemic circulation causes the lungs to be the most natural seat for all forms of *secondary* growth whenever infection takes place by the blood-current, excepting in those cases in which the primary tumour lies within the area of the portal system of vessels. But in very many instances, the patient, up to the time of his death, shows no indication of anything more than a general cachexia, and it is only at the autopsy that the lungs are found studded with nodules or masses of new-growth. On the other hand, there are some cases in which cough and dyspnœa, or hæmoptysis, draw attention to the state of the lungs; and in which there are physical signs of the exact position of one or more tumours, besides other signs of fluid-effusion into one or both of the pleural cavities.

Hitherto I have been alluding only to cases in which the breast or the stomach or some other part is known to be affected with a primary malignant growth. But the analogy of other viscera in which secondary tumours are apt to appear, such as the liver and the brain, would lead one to expect what occasionally does occur; a patient dies of the effects of growths in the lungs, and it is discovered for the first time at the autopsy that these growths were secondary to some primary growth in a distant organ. Such an occurrence, however, appears to be very rare. The only case that I remember is that of a young man who came into Guy's Hospital under Dr Frederick Taylor with what appeared to be acute bronchitis, and died in a few days; at the post-mortem examination it was found that the lungs were full of sarcomatous masses, secondary to a like affection of the testis.

As to *primary* growths in the lung, even less can be said about them than about secondary growths. Cases in which there is only a single mass of considerable size, or in which one among several masses is obviously older than the rest, have often been described as examples of primary malignant tumour in the organ. But I feel uncertain whether the other viscera, and especially the different mucous membranes, have always been searched with sufficient care to justify the conclusion. The same doubt, I think, applies to cases which have been recorded under the name of disseminated or miliary cancer of the lungs. One such I myself brought before the notice of the Pathological Society in 1866. The patient was a man, aged fifty, who died of an illness of two or three months' duration, but only two days after being admitted into hospital, with what appeared to be capillary bronchitis complicated with some pneumonia. At the autopsy the lungs were found full of round bodies like tubercles, but larger (some as large as hemp-seeds) and of a shining white appearance. The only growths discovered elsewhere were a few in the heart and in the liver. A somewhat similar case, in a girl of seventeen, is related by Risdon Bennett;

but the liver in that instance contained several very large tumours, so that the lung affection was clearly secondary.

A still more remarkable form of disease is one of which I met with an instance in 1870. A man, aged thirty-six, died in Guy's Hospital after two and a half months' illness, which was attributed to damp and cold, and which appeared clinically to have been pneumonia of the right lung, accompanied with much effusion into the pleura. At the autopsy I found the lung much enlarged, nearly white in colour, but somewhat mottled, smooth, and shining in appearance, soft and cushiony to the feel, so that one might have imagined it to be generally emphysematous but for the fact that it was absolutely airless, every part of it sinking instantly when put into water. At the root of the lung there was obvious new growth, which probably was seated in the glands, but which had also involved the superior vena cava and narrowed it considerably. Unfortunately, the lung was thrown away before any microscopical examination of it was made. But my impression is that it was an example of a primary diffused carcinoma of the pulmonary tissue, such as is alluded to by Hertz (in Ziemssen's 'Handbuch') as "bearing a striking resemblance to grey hepatisation."

Thrombosis and Embolism.*—Thrombosis implies a spontaneous coagulation of the blood in a blood-vessel during life, and embolism the carrying away of some detached fibrinous material to a distant vessel which thus becomes blocked.

As regards thrombosis, coagulation may occur under various conditions and from numerous causes. Anything which tends to render the blood stagnant will favour its coagulation, and afterwards certain changes take place in it; but if the blood be flowing during the formation of a thrombus, then the fibrin is deposited, portion by portion, until a white fibrinous mass fills the vessel. The principal cause of coagulation is some change in the interior of the vessel; whilst this is smooth and in a state of vital integrity the blood remains fluid, but as soon as the endothelium is in any way altered in structure, or any part of it removed, or cretaceous material be formed upon it, coagulation will take place. The effect of retardation of the blood-current is seen in the case of coagulation in the iliac veins of young persons dying of phthisis and other wasting disorders, and the effects of disease in thrombosis of the vessels of the aged brain. There are no doubt other causes which favour coagulation inherent in the blood itself; the physiologist speaks of various special states which promote it, and probably like morbid changes may occur in various dyscrasiæ and determine its coagulation. For example, coagulation of the blood leading to thrombosis is often met with in diabetes, gout, typhoid, the puerperal state, and other disorders. In these cases a coagulation occurs in a small vein, and then proceeds upwards, sometimes reaching to the vena cava.

The clot may set up an irritation or inflammation of the lining of the vessel, so that there is soon associated with the coagulum an endarteritis or endophlebitis. The clot then becomes organised and the vessel obliterated. Occasionally it may shrink and allow a passage of blood through it again. In other cases the clot softens and is changed into a milky or pus-like material.

Blood-vessels may become independently inflamed. Thus the intima of an artery undergoes inflammatory processes just like those of the endocardium, leucocytes may be seen infiltrating its walls, and finally fibroid patches form on the surface which may terminate in sclerosis and calcification, or the vessel become merely thickened. Veins in connection with local inflammations may suppurate, as is often seen in the jugular in cases of disease of the temporal bone or in the portal in association with abscess of the liver.

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* [See footnote, vol. ii, p. 68.—Ed.]

Cases of thrombosis become most important when the coagulation reaches the iliacs or vena cava, for then, should a portion of the clot become detached and carried to the heart and so on to the pulmonary artery, almost sudden death ensues. The most common and best marked instance of this kind is that where a clot has formed at the commencement of the vena cava as an extension from the iliac veins in puerperal cases. Some inflammatory process having occurred about the neck of the womb, coagulation takes place in the uterine sinuses and extends through the iliacs to the vena cava. This may have occurred with so very few symptoms that no suspicion of the coming fatal event is excited. It is usually about a fortnight after delivery, on the patient's rising from bed, that a portion of the fibrinous clot becomes detached, passes into the pulmonary artery, and causes almost instant death. If the clot should be small and plug only one pulmonary artery or a branch, then death may not occur so speedily, but be postponed for a hour or two. In other cases the detached portions are still smaller, and not being large enough to close the vessel entirely, they stick in some of the branches, and there form a focus for further coagulation. This proceeds until the whole lung is blocked, and a fatal termination may be delayed for a few days. Under these circumstances the patient, though very rarely, may recover.

With the exception of the case of pulmonary embolism just mentioned, the most important and frequent forms of embolism are those which are met with on the arterial side of the system. A clot may form, for example, in a large artery and portions of this may be carried into the vessels beyond, or, what is more common, vegetations and portions of fibrin become detached from the valves of the heart and are then carried into the arterioles of various parts of the body. For example, there is the sudden blocking of the middle cerebral artery, a very common cause of hemiplegia, or of the vessels of the limbs leading to gangrene. Several cases of this kind have occurred in Guy's Hospital, as for example, where a boy under treatment for heart disease was suddenly seized with pain in the arm, which on examination was found to be pulseless. Or where a man was suddenly attacked with most excruciating pain in the leg, which on examination was found to be cold and with no pulsation in the arteries. It is remarkable, as before observed, that blood-vessels which are regarded as insensible organs should evince the most acute sensibility when subject to lacerations or to stretching such as occurs in embolism.

One of the subsequent events which occasionally occurs in the vessels so blocked is the formation of aneurysm (*vide supra*, p. 69).

When smaller particles of fibrinous matter are detached and carried by the circulation to all parts of the body, as in the so-called ulcerative endocarditis, local effects are not so manifest, but the blood becomes infected, symptoms of a typhoid or pyæmic character are set up, and the malady, if fatal, may be protracted through several weeks. In these cases the arterioles in various organs become blocked, leading to very characteristic formations in the organs, known as "infarctions." The vessel having become blocked and the blood flowing back into the emptied tissue coagulation occurs in these arterioles, and if an exudation takes place from them a mass resembling a portion of fibrin is formed. Such wedge-shaped masses are met with at the edges of the lung, kidney, or spleen in the midst of which the remains of the tissue of the organ may still be seen. These may soften, leading to further infection; or they may dry up, leaving a cicatrix; or in the case of the lung they may become a focus for an inflammatory process.

Cases of ulcerative endocarditis have elsewhere been described (vol. ii,

p. 46), and instances of sudden death from pulmonary embolism need no illustration.

Cases, however, of slow coagulation in the lungs with more lingering symptoms are not so common, and therefore an example of this may be given.

A medical man, fifty years of age, was seized on the 3rd of March with difficulty of breathing, and on the following day he was carefully examined by Dr Wilks. His apnœa was of that kind so frequently seen in cardiac disease; the patient was panting and breathless, the respiration was very quick and on the slightest exertion it almost ceased, as happened more than once on his attempting to move out of bed. On examination of the chest nothing abnormal could be discovered; the heart-sounds were healthy, its action quiet, and the pulse was 80. The patient had no difficulty in taking a very full breath, indeed he felt, he said, as if he must breathe too much. A suspicion of embolism caused an examination of the whole body, when it was found that he had had phlebitis of one leg, arising from an injury six months before. The patient also said that a month ago he was seized with difficulty of breathing, which passed off after a few hours. Dr Wilks concluded from this that he had embolism of the pulmonary artery. On the following day he was no better, and on March 6th I saw him, but could discover no disease in the chest from the existence of any physical signs. On March 7th he was worse and was beginning to spit up tenacious, rusty, and bloody mucus. On March 8th he was dying, gasping for breath and with great lividity of the extremities. The heart was regular and there was no bruit. The autopsy showed embolism of both branches of the pulmonary artery. In the left was a large clot, tolerably recent, and in the right an older one adherent to the walls of the vessel. There was also pneumonic consolidation in portions of both lungs. The right femoral vein contained a thrombus of exactly the same character as that found in the pulmonary artery.

Cases of recovery from pulmonary embolism are so rare that it may be also advisable to give an illustration of it.

A young man, an officer in the army, was operated on for varicocele; he rapidly recovered from the operation, and had left his bed, but had not gone out of the house. During conversation one evening, February 9th, a fortnight after the operation, he suddenly called out, fell back, and gasped for breath. It was thought he was dying. A medical man saw him soon afterwards and found him cold, pulseless, and breathing heavily. About an hour afterwards he spoke; he was very pale, and had sighing respiration, but it was thought that his pulse was perceptible. He lay all night very quiet and calm, but pale, and the pulse was only just capable of being felt. On the following morning he had rallied, his skin had become warm, the breathing tranquil, and the pulse 100. February 11th.—Slight oppression of breathing with little pain; temperature normal. February 12th.—Slight crepitation heard over lower part of chest on left side. Heart normal, no bruit. February 13th.—Lying quietly, filled chest well and deeply. Crepitation over lower part of left chest in front and expectoration of a little bloody mucus. A slight murmur heard at base of heart. February 14th.—Bruit more audible and traced up left side in course of pulmonary artery. February 16th.—Bruit less marked and crepitation of lung less. February 20th.—Bruit gone and no râles heard, but in the spot where they existed imperfect respiration. The expectorated stuff was peculiar, being a glairy mucus, interspersed with streaks of blood. He continued to improve, but still spat up red, glairy, transparent mucus, and when he sat up, the pulmonary bruit became audible. March 15th.—Allowed to leave his bed, all physical signs having disappeared from the chest, but he still spits a little coloured mucus. He was kept quiet for another month, when he was allowed to go out, and gradually recovered.*

Exophthalmic Goitre.—In the second quarter of the present century several observers noted the concurrence of palpitation of the heart with staring eyeballs and an enlarged thyroid body. In Germany such cases are now commonly spoken of as examples of "Basedow's disease," because v. Basedow in 1840 published a paper on the subject. It had, however, already been mentioned by Dr Graves, of Dublin, in his 'Lectures' (originally published, according to Dr Stokes, in 1835); and therefore there is some warrant for calling it "Graves' disease," as was proposed by Trousseau. But still earlier allusions to it have been found in the writings of Adelmann, Caleb Parry (1825), and Flajani (1798). Perhaps the best name is that of "Exophthalmic goitre."

Origin.—The complaint generally sets in very gradually; as a rule no definite cause can be found for it. But sometimes it has been traceable to a severe mental shock, and writers say that it may then develop itself rapidly. Trousseau speaks of a lady who, having one night been crying for a long time on account of her father's death, "suddenly felt her eyes swell and lift up her eyelids," while at the same time she had copious epistaxis, violent palpitation of the heart, and throbbing and enlargement of the thyroid; four days later she consulted a medical man, who recognised the nature of the case.

It occurs chiefly in young women, but a patient who died of it at Guy's Hospital in 1868 was a woman of fifty-eight, and Dr Stokes saw it in a lady upwards of sixty years of age. It is said to have been observed in a girl of seven, and in another girl only two and a half years old. The proportion of females to males is stated by Eulenburg (in Ziemssen's 'Handbuch') as two to one; but Trousseau gives it as fifty to eight, which certainly seems to accord better with the impression that I had formed from my own experience.

Persons who are anæmic or chlorotic are said to be especially liable to exophthalmic goitre; and Stokes mentions the case of a man in whom long-continued bleeding from piles was supposed to have caused it. It also frequently occurs in women who are hysterical, and in neurotic subjects, including epileptic patients and lunatics. An instance is recorded of it in a boy, aged eight, whose mother is said to have had the same disease. And a man, aged twenty-six, whose portrait was given by Dr Wilks in the 'Guy's Hospital Reports' for 1870, had an uncle and a brother who were affected with ordinary goitre.

Symptoms.—Of the three cardinal symptoms, the first to be observed is commonly an *increased action of the heart.* This may for a time be only occasional, but afterwards it is constant. The number of beats is much augmented, reaching 120, 140, or even (it is said) 200 in the minute. The cardiac impulse is exaggerated, the sounds are loud and ringing, and a blowing systolic murmur may be audible with the stethoscope at the base or even at the apex. The carotid arteries throb, and with the hand one may feel pulsation of the enlarged thyroid body, or a well-marked thrill. The beats of the radial arteries, however, are not unduly forcible. Trousseau, in opposition to Aran, makes it a point that there is no increase in the area of absolute cardiac dulness, and I can confirm his statement from my own observations. This would not of itself prove that the heart may not be enlarged (see vol. ii, p. 24). But in the fatal cases that have occurred at Guy's Hospital, the organ has weighed only nine, ten, or once perhaps eleven ounces. It is true that each patient was much emaciated, so that a degree of relative hypertrophy may be said to have been present. Atheroma of the aorta has been mentioned as an effect of the increased strain upon that vessel.

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* [I have met with two cases of recovery from pulmonary embolism; one in a patient I saw with Mr. Hine, of Oxford, where it followed thrombosis of the femoral vein from an injury; the second, during recovery from pneumonia, in a healthy man who was under the care of Dr. J. H. Galton. In both cases the condition was recognised before I saw the patient, and in both the diagnosis was amply confirmed.—Ed.]

Swelling of the thyroid body may either be observed before the exophthalmos, or not until after that symptom has attracted attention. It is sometimes symmetrical, sometimes more marked on one side, and usually on the right. In some cases it is but slight, so as hardly to deserve to be called a goitre, and I believe that it never approaches in size the very large tumours which sometimes occur in the endemic form of that disease. But it may cause a considerable projection of the throat, and it may press on the trachea, so as to alter the voice and to compel the patient to lie with the head thrown back upon a pillow. In Dr Wilks' case, already referred to, the lower part of the left lobe was found after death to extend down into the chest, altering the shape of the trachea, and perhaps compressing the thoracic duct. In an early stage of the disease the thyroid body sometimes undergoes rapid variations in size, becoming larger when the heart's action is more disturbed, or even under emotional excitement on the part of the patient. This of itself shows that the state of the gland is partly one of vascular turgescence, and its vessels have been found increased in diameter, the arteries especially being tortuous. Its tissue may be perfectly normal in appearance, or it may contain more or less numerous "colloid cysts," some of which may be of considerable size.

Prominence of the eyeballs varies greatly in degree; one patient merely appears to stare a little more than natural, another has a fierce and savage expression, there being a wide space between the corneal margin and the edges of the eyelids. It is even said that the points of insertion of the recti muscles into the sclerotic may be visible; and Trousseau alludes to a case in which one of the eyes "actually came out of the orbit, and had to be pushed back by the fingers." Sometimes the protrusion is more marked or begins earlier on one side than on the other; according to certain observers it may be permanently unilateral. A point to which von Graefe attached considerable diagnostic importance is, that when the patient looks downwards towards the feet the upper eyelid fails to descend, by an associated action, as it normally should do. He showed that this is by no means a mere necessary consequence of the exophthalmos; for when a tumour of the orbit causes the eye to project the movements of the eyelid remain unimpaired. In one patient whom he saw, and whose complaint was of palpitation of the heart, the symptom in question constituted the sole ground for regarding the case as one of Basedow's disease. On the other hand, Eulenburg says that he has found "von Graefe's symptom" absent, or almost absent, when there was great protrusion of the eyeballs. In cases in which there is a high degree of exophthalmos, the eyelids may fail to meet during sleep; the cornea is then apt to become inflamed and even to slough. As a rule, patients affected with exophthalmic goitre see perfectly well, but sometimes they notice muscæ, or complain of fatigue in using the eyes. The only ophthalmoscopic appearance is said to be a dilated and tortuous state of the retinal veins. The pupils have been described as being generally dilated, but Eulenburg cites von Graefe as having failed to observe this in an experience extending over nearly 200 cases, and he suggests that when it has been present it has been accidental and due to myopia. There appears to be still a doubt as to whether the cause of the protrusion of the eyeballs is turgescence of vessels in the orbit, or an overgrowth or swelling of the fat. A third hypothesis is that it may in part be due to contraction of Müller's non-striated orbital muscle. Exophthalmos has sometimes been noticed to increase or diminish as palpitation became more or less severe, and to vary with the menstrual periods. In some cases, but not always, the eyes are scarcely, if at all, prominent in the dead body. The recti muscles have twice been found in a state of fatty degeneration, but this is attributed to their having been disused or to the stretching which they had undergone.

Other symptoms which have been noticed in patients suffering from exophthalmic goitre are irritability of temper, sleeplessness, headache, impairment of memory, unfitness for employment, voracity of appetite, flatulence, constipation, amenorrhœa, leucorrhœa, epistaxis. Irregular febrile attacks sometimes occur, in which the temperature may rise 2° or 3° F. There is often extreme emaciation. Trousseau notes that he has obtained a *tache cérébrale.* The spleen may be swollen. Enlargement of the breasts has been mentioned, but Trousseau speaks of them as undergoing atrophy. It is perhaps worthy of note that in two fatal cases at Guy's Hospital, the patients being respectively twenty-nine and twenty-one years old, the thymus was persistent. In one it was four inches long, and had a maximum thickness of three quarters of an inch.

Pathology.—I need not discuss at any great length the many speculative views that have been advanced to account for the phenomena of exophthalmic goitre. That no one of the three cardinal symptoms can be taken as the cause of the others is now, I think, certain. Some of the earlier writers on the subject held that a heart affection constituted the starting-point of the disease. Stokes, for instance, believed that this was usually a persistent functional excitement of the heart; and he cited Dr Parry's cases as showing that organic lesions might occasionally be followed by similar effects. Nor must I omit to mention that in two fatal cases at Guy's Hospital there had been an antecedent attack of rheumatic fever, and that in one of them pericarditis and endocarditis were found at the autopsy. On the other hand, Kœben imagined that the goitre gave rise to the rest of the symptoms by pressing upon the sympathetic ganglia; but such a notion is altogether inconsistent with the fact that enlargement of the thyroid may be absent, or may follow the exophthalmos; moreover, the much larger endemic goitres produce no such consequences. There seems, therefore, to be no escape from the conclusion that the phenomena of the disease are joint effects of some other cause; and a very obvious suggestion is that of Trousseau, according to which they are due to disturbance of the lower cervical ganglia of the sympathetic nerves. Eulenburg cites eight autopsies, in each of which changes in their structures were demonstrated; and Dr Goodhart's case in the 'Path. Trans.' for 1874 may be added to them.* On the other hand, there are two cases—one of them investigated with great care by Ranvier—in which the ganglia were found healthy. The changes observed consisted generally in an overgrowth of the fibrous capsules of the ganglia, with or without an excess of the connective tissue in their interior, rendering them hard and tough. And it is perhaps worthy of notice that, in Dr Goodhart's case, the connective tissue of the neck and thorax also appeared to be in excess. In very few cases is it stated that there was atrophy or any other morbid condition of the nervous elements of the ganglia. But if we now attempt to consider in detail how the symptoms of exophthalmic goitre can be caused by lesions of the cervical sympathetic nerves we meet with great difficulties. Boddaert succeeded in producing an enlargement of the thyroid body by ligaturing the jugular and thyroid veins in animals; and when he also divided the sympathetic nerves, the eyeballs became prominent. Thus it may be that paralysis of the nerves in question, by dilating the blood-vessels, gives rise to like effects. But it is worthy of notice that the recognised phenomena of paralysis of the cervical sympathetic are absent. Moreover, the excited action of the heart corresponds not with paralysis, but with irritation of these

* [Also Dr. Shingleton Smith's, 'Med. Times and Gazette,' 1878.—Ed.]

nerves. Some writers have indeed endeavoured to account for all the phenomena of the disease on a theory of "irritation." But it is a sufficient objection to such a view that a primary irritation of a nerve-centre, lasting for months or years unchanged, is as yet unknown in pathology. Lastly, the suggestion has been made that the original starting-point of exophthalmic goitre may perhaps after all be from the spinal cord, or the bulb, or the brain.

Event.—Exophthalmic goitre is not often traced to a fatal termination in hospital experience. We have indeed made six or seven autopsies at Guy's Hospital since 1868; but the only case in which death appeared directly due to the disease was that of which Dr Wilks has recorded the details; and in this there was bronchitis with expectoration of mucus tinged with blood. Two (or perhaps three) patients died of pleurisy, with or without pneumonia; one of rheumatic pericarditis and endocarditis. A woman, who was under my care, succumbed very unexpectedly, after having been ailing for a day or two; the stomach and intestines were found to be affected with a remarkable form of follicular inflammation, the solitary glands and Peyer's patches being very prominent, and the whole mucous membrane intensely injected, swollen, ecchymosed, and lined with mucus. In the remaining case death was caused by the inhalation of an anæsthetic.

In all but two of these cases the disease was of recent origin, having lasted only a few months. One patient had had it for four years. It does not seem that the sufferers from exophthalmic goitre, like those who have other chronic maladies which are incurable, go about from hospital to hospital, until at last they die. And the only possible inference seems to be that most cases at length end in recovery. As yet, however, I know of no positive evidence in support of this conclusion. The duration of the complaint is probably too long, and the natural process of cure is too gradual, to allow of its being followed out in a ward of which the inmates are constantly changing.

Little used to be said with regard to the *diagnosis* of exophthalmic goitre. But it is important to note that one must be on the look-out for slight cases and for rudimentary forms in which one or two of the cardinal symptoms may be absent. This is a point on which Trousseau especially insisted; and Dr Wilks has recorded several instances which might have been set down as examples of ordinary chlorosis but for the failure of ferruginous medicines to cure them.

Treatment.—The medicinal treatment of exophthalmic goitre is indeed very unsatisfactory. Trousseau was strongly convinced that iodine was generally injurious, although he admitted that it was sometimes useful. He also believed that the tincture of iron did harm, and I think that all observers will at least admit that it seldom, if ever, does good. Traube, however, is said to have given iron and quinine alternately, each for three weeks at a time, with great advantage. I have not seen much result from the action of digitalis, which was recommended by Trousseau. In two cases I prescribed ergot, but there was no marked benefit from it.

Some German physicians have recently applied galvanism to the neck—according to the process, so-called, of "galvanising the sympathetic nerves"—with success. A battery of six or eight cells is used; the negative pole is placed upon the spine below the fifth cervical vertebra; the positive pole at different levels in front of the sterno-mastoid muscles. In this way the pulse appears to have been brought down to 70 or 80 from 130, and the general condition of the patient is said to have been much ameliorated. The application of a very full current to the closed eyelids is also recommended as tending to diminish the exophthalmos. In one case, however, I adopted the treatment with but little advantage.

DISEASES OF THE ALIMENTARY TRACT

AFFECTIONS OF THE NOSE, MOUTH, AND SALIVARY GLANDS, OF THE FAUCES AND ŒSOPHAGUS

NASAL CATARRH OR CORYZA.—*Its symptoms, predisposing causes and treatment.*

SUMMER CATARRH OR HAY FEVER.—*Onset and course—Ætiology—Distribution among persons and places—Treatment.*

OZÆNA.—*Origin in atrophy of the nasal tissues—Minor forms—Treatment.*

EPISTAXIS.—*Ætiology—Course—Treatment.*

STOMATITIS.—*Aphthous ulceration of the mouth—Ulcerative stomatitis—Thrush, or false aphthæ—the fungus—Treatment.*

MUMPS.—*Ptyalism—Metastatic parotitis.*

ANGINA.—*Acute tonsillitis or quinsy—Chronic tonsillitis—Granular pharyngitis—Inflammation of the vault of the pharynx, with adenoid growths.*

STRICTURE OF THE ŒSOPHAGUS.—*Minor disorders—Spasmodic stricture—Regurgitation—Simple stricture—Cancerous stricture—Anatomy—Course—Treatment.*

In the present chapter will be described certain affections of the nose, mouth, salivary glands, fauces and gullet, which require notice in a work on medicine. It is not, indeed, possible to draw any satisfactory boundary line between these and the surgical affections of the same parts. But for convenience I have deemed it best to include not only such as habitually come under the notice of the physician, but some others of which a knowledge is necessary to him if he is to avoid making serious mistakes in his daily practice.

NASAL CATARRH.—*Coryza.*—*Cold in the head.*—Almost everyone, at least in climates like that of England, is liable from time to time to a "cold in the head." This affection commonly begins with sneezing, repeated again and again. The nose becomes dry and "stuffy," its mucous membrane may be seen to be swollen and injected, there is difficulty in breathing through it, and the nasal consonants *n, m, ng*, cannot be properly pronounced. Or, from the very commencement, there may be a profuse flow of a thin, saltish, watery fluid from the nostrils, so that the handkerchief has to be used almost uninterruptedly. Smell and taste may for the time be completely abolished. If the frontal sinuses are involved there is dull pain in the forehead, or even severe headache, and (it is said) drowsiness. Extension to the lachrymal passages may cause the tears to flow over the cheeks, and may even lead to the development of a secondary conjunctivitis. Extension to the fauces and Eustachian tube may give rise to deafness. The skin near the nostrils often becomes inflamed and excoriated, a result which is attributed to the "acrid" character of the discharge. In this fluid leucocytes appear to be always present, though sometimes only in small numbers; and, according to Hüter, micrococci are also abundant.

Sometimes an attack of nasal catarrh passes off as suddenly as it began. In two or three hours the flow may cease entirely; or, the person having been tormented with a "running cold" up to the very moment of going to bed, may forthwith fall asleep, and on waking in the morning may find himself entirely free. According to Trousseau, such a transitory coryza is sometimes an irregular manifestation of asthma, either leading directly to a genuine attack of that disease, or at least occurring in individuals subject to it. In cases of this kind there is little or no constitutional disturbance. But the more common kinds of nasal catarrh often set in with marked malaise and chilliness, and in children they may be attended with considerable pyrexia. The usual duration of such an affection is from two days to a week, but sometimes it lasts considerably longer, or (it would perhaps be more correct to say) a series of attacks occur in succession, a fresh one setting in each time as the other is passing off. Whenever coryza runs on for more than a day or two, the discharge from the nose alters in character, becoming viscid, opaque, and muco-purulent. Sometimes, as the nasal affection subsides, the larynx and the trachea are in their turn attacked with a like catarrhal inflammation. Lastly, a severe cold is in certain cases followed by a long-continged impairment or loss of the sense of smell; indeed I believe that this may sometimes be permanent.

In regard to the *causes* of coryza, it is in the first place to be noted that some individuals are far more liable to it than others, and that it is also especially apt to occur in certain families. There are persons who are sure to take cold if they get their feet wet; but such an over-susceptibility to catarrh may often be controlled by cold sponging in the morning, by the regular use of a shower-bath, by keeping the bedroom supplied with fresh air during the night, or by other like measures. Among the public it is almost universally believed that nasal catarrhs are contagious; they are supposed to be easily conveyed from one person to another by handkerchiefs, or even by the act of kissing. The most suggestive medical observation that I know of as tending to support this notion is the statement made by Fränkel (in Ziemssen's 'Handbuch'), that he has repeatedly seen a newly-married man, who had never before had a cold, attacked without any other obvious cause shortly after the wife, she having before been subject to the affection. On the other hand Friedreich is said to have inoculated his nasal mucous membrane with discharges from persons suffering from coryza without any result. It is as yet uncertain whether the very great prevalence of coryza at certain seasons of the year is to be regarded as constituting a real epidemic, or merely as a consequence of the widespread distribution of the common exciting causes of the affection.

As a special variety of nasal catarrh must be mentioned that which arises in certain individuals when they are taking iodide of potassium medicinally; this is supposed by Fränkel to be due to direct irritation of the lining of the nose by iodide dissolved in the mucous secretion. One kind of coryza will be presently described under the name of "hay fever" and another was included under "Influenza" in the first volume. And it must not be forgotten that this affection is a prominent early symptom of measles, and sometimes of typhus.

Again, an inflammation of the nasal mucous membrane that may be mistaken for an ordinary catarrh, is sometimes dependent upon an infection of the nose with leucorrhœal or gonorrhœal discharge. In such cases, however, the exudation is from the first yellow and purulent, or mixed with blood, and the duration of the affection is much more protracted, lasting several weeks. Dr Hermann Weber, in vol. xliii of the 'Med.-Chir. Transactions,' seems to have first drawn attention to the possibility of the nasal cavities of an infant becoming infected during birth when the mother has leucorrhœa.

Fränkel thinks that such an occurrence is by no means infrequent. To obviate it one might in suitable cases employ an injection of some antiseptic fluid just at the commencement of the second stage of labour. In the 'Lancet' for 1857 Mr A. M. Edwards relates a case of nasal gonorrhœa in a woman who was ultimately shown to have caught it by using a handkerchief that had been employed as a suspensory bandage by her son, he having the disease.

Coryza is not a disease about the treatment of which one is often consulted, as most people manage their attacks themselves independently of any medical advice. It is right to keep indoors, or even in bed, according to the severity of the "cold." At the commencement, a dose of Dover's powder may be taken at bedtime with a basin of hot gruel. A warm bath is also useful; setting up free perspiration seems often to favour the rapid subsidence of the affection. Locally, I have seen marked relief follow the insufflation of a bland powder containing bismuth mixed with a little morphia, as suggested by Dr Ferrier ('Lancet,' 1876, vol. i, p. 523).

When nasal catarrh, instead of subsiding, persists for weeks together, change of air is often the best remedy; but I have sometimes seen the internal administration of arsenic very useful under such circumstances, and I have also obtained striking results from the same medicine in some cases in which loss of taste and smell as the result of coryza had continued for many months; in another case, however, it entirely failed.

HAY FEVER.—*Catarrhus æstivus.*—At the present time it is matter of common knowledge that certain persons are liable every summer to a very troublesome affection, which sometimes assumes the character of catarrh, sometimes of asthma. Attention was first directed to it by Dr Bostock in a paper read before the Royal Medical and Chirurgical Society in 1819. Of the *asthmatic* form I have spoken in the first volume.

The catarrhal form sets in with a feeling of irritation in the nose, throat, and eyes. Then the patient begins to sneeze—perhaps twenty or thirty times in succession. A thin watery secretion pours from his nostrils. The nasal submucous tissue rapidly swells, until in a little while no air can be drawn through the nose. If, however, he should lie down and turn on his side the nostril which is now uppermost becomes in a short time free (apparently as the result of gravitation of the œdematous fluid), while the other one becomes more occluded than before. The swelling affects the lachrymal passages also, so that the tears run down over the cheeks. The eyes become inflamed; there may even be œdema of the eyelids. So distressing are these symptoms that it is almost impossible for the patient to avoid giving way to them and suspending his usual occupations. From day to day they vary in severity, but they commonly last three or four weeks, or even longer. Paroxysms of sneezing continue to recur from time to time. The nasal discharge presently becomes thicker and more purulent, or it may be stained with blood. Ultimately the affection passes off, leaving more or less weakness and prostration behind it.

One of the first points observed about this "summer catarrh" was that its onset often corresponded closely with the beginning of the hay season, and that persons were attacked immediately after being in or close to a hayfield. But it was not until 1873 that Dr Blackley, of Manchester, observed by careful observations and experiments upon himself that the true cause of the affection is the diffusion in the air of the pollen of certain plants, and especially of grasses, which settles upon the mucous membrane of the nose and eyes, and acts as a local irritant. Up to that time some medical writers had attributed hay fever to coumarin (a chemical principle contained in the sweet-scented grass, *Anthoxanthum odoratum*), others to ozone, others to "common dust," others to the mere heat of early summer.

Dr Blackley found that by introducing a small quantity of pollen into the nostrils he could bring on all the symptoms of the disease almost instantaneously. During the summers of 1866 and 1867 he made daily observations upon the amount of pollen which was deposited upon glass slides moistened with glycerine and exposed to the air; and he found that there was in general a close relation between the quantities collected and the severity of the symptoms of hay fever under which he laboured from day to day. Dr Blackley appears to have accounted satisfactorily for certain observations which had seemed to Dr Bostock and others to prove that the supposed connection between summer catarrh and the emanations from flowering plants was a mistake. At any rate he showed that it is often quite possible to imagine that there can be no pollen in the air when it is really present in abundance. Once he was suddenly seized while at the sea-shore, with a sea breeze blowing; between him and the sea there was but a narrow belt of land, but upon this he found a field of wheat in full bloom. Another time an attack was brought on by the dust of an unfrequented country lane; it was summer time, and on examining the superficial layer of dust with the microscope he discovered that it was full of the pollen grains of grasses.

Hay fever most commonly appears for the first time about the age of puberty, but sometimes it is observed in children four or five years old. Persons who have reached the age of forty without being affected with it are probably never attacked afterwards. It is more common in males than in females.

One very curious circumstance about this disease is that those who suffer from it appear always to belong to the educated classes. It is never seen in farmers, who are of course more exposed to the influence of pollen than any other set of men. This fact has led to the suggestion that the individual predisposition to the disease, which plays so important a part in its ætiology, is perhaps a result of an indoors life, especially in towns or cities. Should that be the case, one can understand how it is that hay fever appears to have become so much more common of late years than in the earlier part of the present century.

It is supposed to prevail in England much more than on the Continent. In most cases the susceptibility to the disease appears to increase with each successive year. At first the patient may be attacked only when he is actually in a meadow where the grass is in full bloom; ultimately he suffers as soon as he attempts to go into the country during the hay season. Sometimes hay fever begins to assume the asthmatic form only after it has for several years recurred in the catarrhal form.

The *treatment* of hay fever is extremely unsatisfactory. Neither quinine,* nor arsenic, nor any other medicine appears to have the power of enabling those who are liable to the disease to bear exposure to its exciting cause without being at once attacked by it. For those who suffer severely, the only course is to remain in a large town through the whole of the summer months, or else to go to the seaside, choosing some narrow peninsula or island, or to take a sea voyage. Staying indoors during the middle of the day, even in a country house, often does a good deal to mitigate the symptoms. It is to be understood that the continuance of hay fever during several weeks—generally from some time in May until the middle of July—is simply dependent upon the continued exposure of the patient to the exciting cause of the disease. If he can get away to a place where there is no pollen in the air, the attack quickly passes off. When the symptoms are first coming on I have seen considerable relief afforded by the use of a smelling bottle containing ammonia, iodine, and carbolic acid, made into a paste with wood-charcoal, and compound tincture of benzoin.

* [The local application of quinine in solution was first used in his own case by Professor Helmholtz, but it has not proved so successful with meaner sufferers.—Ed.]

Ozæna.—A very intractable and distressing affection is one in which the air expired through the nose has constantly a peculiar and disgusting fœtor: this is known as "ozæna."

With regard to many points about it there is still much uncertainty. Formerly it was thought to be generally dependent, either upon a scrofulous diathesis, or upon a syphilitic taint, congenital or acquired. There is of course no doubt that disease of bone in the interior of the nose, whether due to syphilis or to any other cause, may produce great fœtor. But the better opinion seems to be that the special odour that characterises ozæna is not generally traceable to such lesions, nor even to ulceration of the mucous membrane. All recent observers who have made autopsies in cases of ozæna appear to be agreed that in this affection there is an *atrophic* condition of the tissues within the nose, including even the turbinated bones. At one time it was supposed that this was the ultimate stage of a chronic "rhinitis," attended at an earlier period with thickening and swelling of the mucous and submucous structures. There is, however, no evidence of the occurrence of such an antecedent condition. Nor is it clear how the atrophy, when it has developed itself, is related to the fœtor. Some have thought that the current of air through the widened nasal passages, being unduly slow, fails to clear away the mucus which is constantly being secreted, and which therefore dries up and undergoes decomposition. Others hold that the wasted condition of the glands of the mucous membrane leads to an insufficient formation of mucus. In either case it appears probable that the process of desiccation is essential to the production of the offensive odour. But, on the other hand, there can be no doubt that mucus may and often does form dry crusts in the interior of the nose, without any fœtor resulting. The probable explanation seems to be that for the production of ozæna a special ferment is required, the presence of which induces a particular kind of putrescence.

So penetrating is the smell in the worst cases of this distressing malady, that it pervades the air for some distance round the patient, and renders him unfit for society and for duties by which he would be brought into contact with other persons. Yet he himself is often entirely unconscious of it.

It must not be supposed, however, that the affection is always so severe as this. Fränkel remarks that about some persons, when they first wake in the morning, very faint indications of the ozæna-odour can be plainly recognised, which are altogether absent at other times. And, again, he says that in some cases in which mucus leaves the nose free from smell, it acquires more or less of the characteristic fœtor in drying on the handkerchief. Both these statements I can confirm.

Until recently, the usual method of treating ozæna has been by systematic irrigation of the nose with water, or with saline solutions, or by injections of antiseptic agents, such as boracic acid or salicylate of soda. Of late, however, a considerable advance seems to have been made by Gottstein, of Breslau, who simply introduces a plug of cotton wool into one nostril, leaving it there for twelve or twenty-four hours, and then withdrawing it, and plugging the other nostril in its turn for a like period. The effect is often completely satisfactory so far as concerns the temporary removal of the fœtor. But almost as soon as the treatment is at any time discontinued the case becomes as bad as ever. Ozæna is, in fact, according to present experience, incurable.

So serious is the discomfort entailed and so unsatisfactory have milder remedies proved, that more than once surgeons have undertaken the severe operation of opening the nostril and extirpating the turbinated bones. (See on this subject a paper by Mr. Warrington Haward in the 'Lancet' for 1877, vol. i, p. 784.)

Epistaxis.—*Bleeding at the nose.*—In various chapters of the present work allusion is made to epistaxis as a complication of other maladies. It is apt to occur in all hæmorrhagic diseases, and in splenic leuchæmia it is often an early symptom. Persons who have granular wasted kidneys with hypertrophy of the heart are exceedingly liable to it, and in them it may often be taken as a warning of the probable supervention of uræmic seizures or of cerebral hæmorrhage. It is also frequent in persons who have cirrhosis of the liver, with partial jaundice and with dilatation of the small veins of the cheeks. It may accompany some of the acute infective diseases, especially enteric fever. In cases of ague not only is it sometimes associated with the ordinary paroxysms of the disease, but it is said to have occurred periodically as the sole symptom and effect of malarial poisoning, until stopped by the administration of quinine.

But in the majority of cases, bleeding at the nose is due to none of these causes. There are some individuals, especially boys towards the age of puberty, who are very subject to it. Not only does it arise in them spontaneously from time to time, but at any moment a slight blow upon the face, touching the nasal mucous membrane, or forcibly blowing the nose, may bring it on. In the 'Lancet' for 1865, Dr Babington gave the history of a family in which a marked liability to habitual and violent epistaxis was traced through five generations, and in thirteen out of twenty or more individuals. Some of those who suffer from recurrent or "habitual" epistaxis are pallid and anæmic, with apparently little blood to spare. But others are plethoric looking, with flushed cheeks and injected conjunctivæ; and they may experience from time to time sensations of oppression and giddiness, noises in the ears, throbbing in the head, fulness and heat in the nose, which they recognise as indications of the approach of an attack of hæmorrhage, and which are at once relieved when it occurs. Lastly, in some young women, epistaxis has been observed to be distinctly vicarious of the catamenia. The most striking instance of this that I know of is one recorded by Obermeier in vol. liv of Virchow's 'Archiv.' A girl of fifteen, after once menstruating in the ordinary way, began to suffer at regular intervals of a month from bleeding at the nose; this occurred two or three times a day for three successive days, and it was attended with malaise and other symptoms like those which had accompanied the natural flux. She became pregnant and the hæmorrhage then ceased, to return six weeks after her confinement. No doubt caution is required in accepting the statements of patients as to any form of vicarious menstruation; but this case at least appears to be beyond question.

In epistaxis the blood almost always comes from one side of the nose only; but sometimes part of it passes round behind the nasal septum and ultimately escapes from the opposite nostril, so that both sides may appear to be bleeding simultaneously. It may either flow in drops or in a more or less continuous stream; the quantity lost is in some cases very great indeed. It is commonly of a bright red colour, this being perhaps due to exposure to the air after it has escaped from the vessels. It forms a solid coagulum. One can very seldom discover, whether by rhinoscopy or by inspection of the nose through the nostril, the exact point from which the oozing of blood takes place, nor have pathological anatomists as yet ascertained whether in cases of habitual epistaxis any of the veins in the submucous tissue of the nose (which are normally very wide and numerous) are in a varicose condition or affected with any degenerative change in their walls. In many cases a part of the blood escapes through the posterior nares into the pharynx and is swallowed. Indeed, if the patient should happen to be asleep or recumbent from any cause, the whole of it may take this course; and when a large quantity of blood is subsequently ejected from the stomach by vomiting, the real seat of the hæmorrhage may be altogether overlooked unless a careful examination of the nose is made, which will almost always reveal the presence of clots in one or the other of the nasal cavities or in the naso-pharyngeal space.

The ordinary course of epistaxis is to cease spontaneously sooner or later. But it sometimes continues for many hours or even for days, without intermission. In such cases the patient may rapidly pass into a condition of extreme anæmia and may possibly die of syncope. And in other cases the frequent repetition of attacks of bleeding at the nose brings about a chronic state of bloodlessness that itself tends to favour the occurrence of further hæmorrhage. The process by which the natural arrest of epistaxis is effected appears to be by the formation of coagula, which adhere firmly to the mucous membrane and close up the vessels. Hence, after the cessation of an attack any disturbance of the parts may at once cause a return of bleeding. Fränkel, in Ziemssen's 'Handbuch,' remarks that obstruction of the free part of the nose by clots, with apparent cessation of the epistaxis, affords no guarantee that oozing into the pharynx may not still continue, the patient swallowing the blood without knowing it; in a case of enteric fever, for example, great danger may arise in this way.

Epistaxis should not always be actively treated; as I have already observed, it may give relief to other symptoms of which the patient had been complaining, and it then generally ceases of its own accord. On the other hand, when anæmia is beginning to result, one must be careful not to delay too long in carrying out whatever measures may be requisite. Sometimes compression of the side of the nose against the septum by the finger placed just below the nasal bone is sufficient to arrest the flow of blood, at least for the time. It is important to notice whether this is the case: for, if so, one can be sure that the bleeding spot is in the fore part of the nasal cavity, and that one can stop all further hæmorrhage by the "anterior tampanode," that is by systematic plugging with a long strip of lint introduced through the nostril. But before adopting this method of treatment it is as well to make trial of astringents, such as gallic or tannic acid, which may be sniffed up in the form of a powder. To inject cold water or a solution of alum or any other astringent liquid is less advisable, because it tends to disturb any clots that may have formed. Cold may be applied to the outside of the nose as well as to the whole of the patient's neck and chest. At the same time he may have his feet placed in a hot mustard bath. He should sit upright with his head slightly bent forward, so as to prevent the blood passing backwards towards the pharynx, and should keep the hands raised above the head.

But if the hæmorrhage should continue, and if the source of it should appear to be from the back part of the nasal cavity, one must not wait long before having recourse to the radical method of the "posterior tamponade" or "plugging the posterior nares." This is generally effected by means of an instrument which is described in every surgical work and which is known as a "Bellocq's canula;" the plug being a piece of folded lint of suitable size and shape. It has, however, its disadvantages; for if the lint is not left undisturbed for some days, epistaxis is apt to begin again as soon as it is interfered with; while, on the other hand, if it be allowed to remain long *in sitû*, there is often great difficulty in loosening it, and I have seen the mucous membrane extensively torn away. In any case it should certainly never be left to become fœtid by putrefaction of the blood and other fluids which soak into it. Of late years the use of Bellocq's canula has been often avoided by filling up the nasal cavity with an elastic pouch, which having been introduced in a collapsed state, is afterwards inflated with air until it exerts considerable pressure in every direction.

Affections of the Mouth.—*Aphthous ulceration.*—Among the trivial affections to which children, or sometimes also adults, are liable, is one which consists in the formation of one or more shallow, round, or oval ulcers in the interior of the mouth. They begin as small, raised, white spots, looking like vesicles. These in a few hours lose their roofs, apparently as a consequence of maceration in the fluid which constantly bathes them. The ulcers which result have an ash-grey or yellow surface, and a bright red border; they are painful, and they are also very sensitive to the contact of particles of food, especially sugar and salt. A favourite seat of them is upon the inside of the lower lip, especially where it joins the gum; they may also occur upon the lining of the cheek or upon the tongue. Some persons are more liable to aphthous ulcers than others, being troubled with them for a length of time at intervals of weeks or months. After a few days such ulcers heal of themselves, and it does not appear that any treatment is necessary.

Single ulcer of the palate.—In marked contrast with this affection is one which is described by Vogel (in Ziemssen's 'Handbuch') as occurring in weakly infants, especially those brought up in lying-in or foundling institutions. It consists in the formation of a flat ulcer at the back of the hard palate, just where the *velum* joins it. There is not usually any tendency to spread deeply, but neither is there any disposition to heal, and the ulcer remains until the child's death, which is usually from diarrhœa.

*Ulcerative stomatitis.—Putrid sore mouth.—Stomacace.**—Under these names is known a disease of considerable gravity, and of which one of the most marked symptoms is a great fœtor of the breath. It is attended with more or less extensive ulceration of the mucous membrane of the mouth. This is especially marked upon the gums, the edges of which become reddened and swollen, are detached from the teeth, and finally seem to break down into a grey pulp. So complete may be the destruction that the sockets of the teeth are sometimes exposed, and the teeth themselves become quite loose and fall out. The whole of the lining of the cheeks and lips becomes the seat of ashy ulceration. The tongue is large and doughy looking, marked at its edges by the teeth, thickly furred or ulcerated on the surface. A large quantity of acid fluid escapes constantly from the mouth, running out upon the pillow while the patient is asleep. All movements of the mouth are very painful, and food is taken with much difficulty.

The chief instances of this affection that I have seen have been in children between the second dentition and puberty; once, I think, two sisters came to me with it at the same time. Among the soldiers of the French army it is stated to be frequent, occurring epidemically when they are overcrowded in close quarters. Whether it is then contagious appears to be doubtful. Bergeron is said to have inoculated himself successfully with it in the lower lip.

The remedy for ulcerative stomatitis is chlorate of potass, which may be given in ten-grain doses at frequent intervals, dissolved in water. Or lozenges containing the salt, or Wyeth's compressed tablets, may be used with the object of securing its local action upon the mucous membrane, which action, however, appears after all not to be so essential as that resulting from its absorption into the blood. It is surprising in how short a time the affection is brought to an end; within three or four days the diseased parts begin to show a clean, healing surface. An example of this action in an adult is given in the 50th volume of 'Virchow's Archiv' (1870), p. 462, where under the title "Gingivitis," three cases of ulcerative stomatitis are related.

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* [A French term—"mouth-ill"—often applied to scurvy.—Ed.]

Thrush.—In this country it has been usual to apply the term *aphthæ* to a condition of the mouth altogether different from that which, in accordance with the universal practice on the Continent, I have called *aphthous ulcerations* (see last page). It is, however, better to avoid such a use of the term, for we have in the vernacular term "thrush" a very suitable name for the affection now to be described; in French it is known as *muguet,* and in German as *Soor.*

The earliest indication that thrush is setting in is a change in the mucous membrane lining the cheeks and other parts of the mouth; it becomes redder than natural, hot, and painful. Soon a number of minute milk-white spots appear upon its surface. These rapidly increase in size, and run together; and in a day or two the whole surface may be covered with a nearly uniform adherent layer. At first there is some difficulty in detaching the white material. But after a time it becomes quite loose, and can be peeled off in large flakes without any bleeding. Microscopically it consists partly of layers of squamous epithelium, partly of the spores and mycelium of a fungus.

This fungus is commonly known as the *Oidium albicans*, although, according to Hallier, it is not really distinct from the *Oidium lactis,* which is the active agent in the souring of milk. It must not be supposed that the presence of the oidium in the human mouth is peculiar to cases of thrush. It has been found upon diphtheritic membranes, and I have seen it in portions of fur taken from the tongue. Nevertheless, I agree with those who think that the essential cause of thrush is a vigorous and rapid growth of this fungus leading to inflammation of the mucous membrane and detachment of epithelium, just as the fungus of ringworm produces a red and scaly condition of a part of the skin upon which it implants itself. There appears, however, to be one thing necessary before the oidium can germinate actively within the mouth. This is an acid state of the secretions which moisten its surface; and according to Vogel, the mouth always gives an acid reaction at the very commencement of the affection, and before any white spots are visible. The preponderance of mucus, which readily turns acid, over the alkaline salivary fluids in young infants is perhaps the reason why thrush is so very much more common in infancy than at any other period of life.

In young children thrush may come and go with but little disturbance of the health, and without any danger to life. But in other cases it is associated with severe and even fatal diarrhœa. There is then a popular notion that the affection passes through the whole length of the alimentary canal, emerging at the anus. But although it is true that the œsophagus is sometimes affected in its entire length, there is no reason to believe that the oidium albicans is capable of germinating on any surface which is not provided with a squamous epithelium. Thus it never enters the nose or the larynx. But it does sometimes appear in the lowest portion of the rectum and upon the female genitals. And it may also be seen upon sore spots on the skin of the face and neck. The relation, therefore, of thrush to diarrhœa in young infants probably is that they are both effects of a weakly state of health from bad feeding or from some other cause. On the other hand in adult life thrush never occurs except in persons who are reduced to a state of extreme marasmus by a chronic malady (such as consumption or cancer) or who have passed through several weeks of pyrexia from some such disease as enteric fever, or puerperal fever, or pyæmia. Thrush in adults is always a sign that the powers of life are nearly exhausted, and it is generally taken as warranting a most unfavourable prognosis; but such patients do nevertheless sometimes recover after having had thrush, if the principal disease from which they suffer is not in itself incurable.

In the treatment of thrush all that is necessary is to wash out the mouth at frequent intervals with a weak solution of an alkaline carbonate or of borax or to apply the *glycerinum boracis* freely to the surface of the mucous membrane.

Affections of the Salivary Glands.—*Salivation* or *ptyalism.*—In patients submitted to active mercurial treatment the mouth is very apt (unless due precautions are observed) to present an affection identical with that which has been described as "ulcerative stomatitis." But in addition there is also an extraordinarily profuse flow of saliva, so that the condition is known as salivation or ptyalism. On the other hand, salivation itself may occur independently of the administration of mercury, being then sometimes a result of the action of other drugs (as iodide of potassium or pilocarpin), sometimes dependent upon irritation starting from distant organs (such as the uterus or the stomach), sometimes apparently spontaneous or idiopathic. But in practice such cases fall into insignificance in comparison with those that are due to the medicinal use of mercury. The quantity of saliva that is poured out is sometimes astonishing. The usual daily amount is one or two quarts; but as much as five quarts are said to have been collected in extreme instances. The patient is incessantly spitting it out of his mouth, or allowing it to dribble forth into a spitpot which he keeps constantly by his side; at night it saturates his pillow. It is more or less viscid or glairy in consistence, and is said to have sometimes a specific gravity as high as 1059; but as the case goes on its specific gravity falls till it is scarcely above that of water. It contains little sulphocyanide of potassium and less ptyalin.

Mercurial salivation is now very seldom seen. In administering mercury one watches the patient's mouth carefully, and the medicine is at once stopped when any disagreeable odour is perceptible in his breath, or when his gums become in the least degree inflamed, or his teeth tender on pressure. Or, if there should be special reason to anticipate the onset of salivation from the fact that one is using mercurial inunction, or calomel vapour baths, or full doses of blue pill instead of minute doses of the bichloride, one may often obviate it by giving at the same time chlorate of potass. When once salivation has developed itself chlorate of potass appears to have little direct influence upon that symptom, although it more or less quickly brings the gums and the mucous membrane of the mouth into a healthy state. The ordinary duration of salivation is from one to three weeks. While it lasts the patient generally undergoes rather rapid emaciation. His urine is scanty and his bowels are constipated. Relief may be given by washing out the mouth with astringent solutions as of alum, or gallic acid, to which some tincture of myrrh and tincture of opium may be added.

Mumps.—*Parotides.*—The most frequent affection of the salivary glands is one which, although it appears like a mere local inflammation, is in reality the expression of a specific disease, transmitted by contagion, occurring epidemically, and possessing the power of protecting against its repetition in the same individual.

For this disease the popular name of Mumps is the best; in French it is known as *Oreillons*, in German as *Ziegenpeter*.

The earliest symptom of mumps is commonly an aching pain in the parotid region on one side, increased by every movement of the jaw, as in speaking, or in taking food. But sometimes malaise and pyrexia precede by a day or two all local signs of the affection. Swelling very soon sets in; the hollow between the mastoid process and the jaw is filled up, its place being taken by an ill-defined projection, which throws outwards the lobule of the ear, and extends over the cheek towards the angle of the mouth, and downwards some distance into the neck. A little later the opposite side usually participates in the disease; and in the course of from three to six days the whole of the face becomes surrounded by an immense mass of firm doughy infiltration, which gives to the patient a truly ridiculous aspect, there being an enormous double chin and the natural contour of the throat being altogether lost. The skin over the affected parts may either be slightly reddened or pale and waxy looking. Internally the swelling extends to the tonsil and the pharynx. The movements of the jaw are greatly impeded. The teeth can with difficulty be separated so as to admit the end of a spoon between them; the patient is obliged to confine himself to a fluid diet, with eggs, custards, and other things that require no mastication.

Sometimes the saliva appears to be very deficient, sometimes to be secreted in natural or even in excessive quantities. The head is kept fixed in one position, with the face directed straight forwards. If, as sometimes happens, the affection remains all along confined to one side, the head is turned rather towards that side. Pain and tenderness continue more or less severe. Sometimes there is hardness of hearing, or the patient complains of shooting pains in the ears or of a continuous ringing sound in them. On about the fourth day the pyrexia ceases. It is seldom of much intensity, although temperatures of 104° are now and then recorded, and typhoid symptoms have even been known to develop themselves.

After three to six days the swelling begins to subside, and its absorption takes place so rapidly that within about an equal period of time it entirely disappears. Thus the whole duration of the disease is rarely beyond one or two weeks. It is sometimes followed by desquamation of the cuticle over the affected parts.

In some cases, however, as the inflammation of the face and of the neck passes off, or even when it has for some days been at an end, the patient, if a male, is attacked with acute "metastatic" affection of one testicle, generally (it is said) the right. The organ becomes swollen and painful, and there is sometimes effusion into the *tunica vaginalis*, with œdema of the corresponding side of the scrotum. After a short interval the other testicle may be attacked in its turn. When this complication occurs there is usually a return of the pyrexia. Trousseau has drawn attention to the fact that the secondary orchitis of mumps is now and then accompanied by symptoms of the most alarming character, though apparently not with any real danger to life. In one instance there was suddenly developed a condition of collapse, in another the patient fell rapidly into a typhoid state. What rendered the diagnosis of the second case even more obscure than it otherwise would have been, was that the initial attack of mumps had been so slight and transient that no notice had been taken of it, and nothing could be learnt about it until consciousness returned. High fever and delirium are sometimes present.

Some years ago I saw a case of orchitis from mumps in which the pulse became extraordinarily slow and remained so for several days; if I remember right, the temperature also fell to a very low point and the breathing was much reduced in frequency.

The inflammation of the testicle usually lasts from three to six days, and then rapidly subsides. Not infrequently, however, it leads to a permanent atrophy of the organ. Urethritis has been noted as a complication. No explanation seems to be at present possible of the liability of mumps to set up orchitis; we can only refer it to that mysterious correlation of distant organs, in their proclivities to disease, of which we find so many other examples in pathology.

In females affected with mumps it is said that the mammæ and the external genitalia sometimes exhibit a like tendency to swelling and inflammation. And this fact is of special interest, because of a case observed by Peter, in which a young woman who had amenorrhœa was several times attacked with parotitis at what should have been her catamenial periods, while on other occasions one of the labia became swollen and painful. According to some text books, the ovaries may be subject of "metastatic" inflammation after mumps. But this seems to be very doubtful.

Mumps is most apt to occur in children from ten to fifteen years old, but it is not uncommon in adults. It is said to be more apt to affect males than females. The secondary orchitis is seen chiefly in boys about the age of puberty and in young men. The contagion of the disease is supposed to be transmitted by the breath. The length of the period of incubation is variously stated by writers; it seems to be generally about fourteen days, but may range from six to twenty-two days.

With regard to the exact seat of the morbid process in mumps there is still some uncertainty. It is certain that the connective tissue outside the salivary glands is largely involved in the inflammatory œdema which must constitute the swelling in the disease; and some writers think that within the glands themselves the structure affected is rather the fibrous stroma which supports the glandular acini than the acini themselves.

Very little treatment is required in cases of mumps. The patient should be kept indoors and out of the way of draughts. As has already been remarked, he can take only fluid food. Fomentations may be applied to the swollen parts.

Metastatic parotitis.—In marked contrast with mumps is an affection of the parotid gland which, instead of being a manifestation of a specific contagious disease, arises as a complication of other maladies, such as fevers, dysentery, or even local affections, such as intestinal obstruction from cancer of the sigmoid flexure, as in a case that I lately saw. This form of parotitis is almost always unilateral. It sometimes subsides without suppuration, but it far more often leads to the formation of an abscess, which may either point behind the ramus of the jaw, or break into the external auditory passage, or burrow down into the neck or upwards in the pterygoid region towards the base of the skull.

The idea has been suggested that this so-called "metastatic" parotitis is perhaps after all the result of dryness of the buccal mucous membrane, leading to an obstructed state of Steno's duct, with decomposition of the retained salivary secretion. But for such an opinion there seems to be no sufficient foundation. It is said that the prognostic importance of parotitis occurring in the course of a fever depends very much on the period of the disease at which it is developed. At an early stage it is of very grave significance; at an advanced stage, or during convalescence, it is a comparatively trifling matter.

Affections of the Fauces.—*Sorethroat.*—Systematic writers on diseases of the throat recognise a great variety of affections of the fauces, all of which are attended with stiff or painful sensations in deglutition, and with more or less marked inflammation of the pharyngeal tissues. Of some of these affections I must give detailed descriptions; but there are others of which a cursory notice will suffice.

Thus "catarrh of the pharynx" is sufficiently characterised by redness and slight swelling of the posterior wall and of the palate, coming on after exposure to cold. In persons who have repeatedly had attacks of catarrh a "relaxed sorethroat" is apt to be of frequent recurrence, the fauces feeling dry and painful (especially in the morning on first waking), but the symptoms all passing off after breakfast, while nothing is to be seen beyond elongation of the uvula, a pendulous state of the palate, and perhaps some dilatation of veins in the mucous membrane.

Then there is an "ulcerated sorethroat" which is particularly apt to occur in nurses, students who are in close attendance on the sick, and other persons weakened by unhealthy hygienic conditions. In this affection small white superficial ulcers are seen on the surface of the tonsils and other parts of the fauces. There is a good deal of constitutional disturbance, the breath is foul and the tongue is furred. Wine, quinine, and tincture of iron are indicated in the treatment, and generally bring the affection to an end in a few days. It is necessary to distinguish from ulcers on the tonsils certain circumscribed whitish-yellow patches, which are really masses of inspissated secretion protruding from the mouths of follicles in their substance. Patients who exhibit these patches often come for advice, complaining of a recent sorethroat; I have found guaiacum very effectual in relieving such cases. Trousseau and others describe a "rheumatic sorethroat," which sets in with extremely acute pain, so that the patient can scarcely swallow a drop of water or even his saliva, but which is remarkably fugacious.

Another faucial affection that has been carefully studied by foreign observers is "herpes of the pharynx." This consists in an eruption of opaline vesicles, sometimes few in number, but sometimes so thickly crowded together that a diphtheritic membrane may easily be supposed to be present. Sometimes it recurs again and again in the same individual; sometimes it alternates with a herpetic eruption on the skin or on the genitals.

Quinsy.—Cynanche.—Acute tonsillitis.—Among the inflammatory diseases of the fauces set up by cold there is one that mainly affects the tonsils; the best designation for it is perhaps the popular name of "quinsy." Dr Mackenzie gives figures, based upon an analysis of 1000 cases, showing that it is far more frequent in persons between fifteen and twenty-five years old than at any other age; it is comparatively seldom seen in children, or in adults beyond the age of forty. Some persons are exceedingly liable to this affection, being attacked by it whenever they get a chill, or sometimes even as the result of slight stomach disorder, or (in the case of women) of menstrual disturbance. In many instances there is habitually a chronic enlargement of the tonsils, and upon this an acute inflammatory swelling from time to time supervenes. Quinsy appears to be rather more common during autumn than at other seasons. It is attended with an enormous increase in size of the tonsil, which forms a red, shining, globular mass, projecting into the fauces, and also distinctly to be felt in the neck at the angle of the jaw. When both tonsils are involved they may come into contact in the middle line, being flattened and even (as Bristowe remarks) ulcerated from mutual pressure. But sometimes one of them is alone affected; and sometimes, just when the inflammation is subsiding in one, the other is attacked in its turn. The uvula, the soft palate, and the pillars of the fauces all partake more or less in the swelling. The patient complains of severe pain in the throat whenever he moves his jaw, and especially in attempting to swallow. The pain may radiate to the ears. The amount of pyrexia often seems disproportionate to the severity of the local affection. Even if there is no actual rigor, the patient experiences alternate chills and flushes of heat, and complains of headache, malaise, and pains in the limbs; the pulse may range from 100 to 120 in the minute, and is full and bounding; the temperature rises to 102°, 103°, or even 105°. The tongue is thickly furred and the breath foul.

I once made an autopsy in the case of a young child who had died in Guy's Hospital during the previous night of suffocation as the result of severe swelling of the tonsils from quinsy. Such an occurrence, however, is exceedingly rare. The disease almost always ends favourably, either subsiding more or less rapidly, or else advancing to the formation of an abscess in the tonsil, which breaks and discharges a thick fœtid pus that is generally swallowed. There is then immediate relief to all the symptoms, the patient almost at once feeling perfectly well. The ordinary duration of the disease is three or four days; but if the two sides of the throat are attacked in succession, and not together, it may be prolonged over a week or ten days. In some cases it is said that suppuration starting from a tonsil extends down into the neck, and burrows until it may even reach the chest. Cases also have been published in which tonsillar abscesses have eroded the carotid artery and given rise to fatal hæmorrhage. And in one reported case suffocation occurred as the result of the breaking of an abscess, which filled the upper air-passages with pus. Dr Mackenzie, who cites this case, remarks that such an accident is much to be dreaded should the abscess give way during sleep.

Some French writers, especially Maingault and Gubler, have maintained that tonsillitis is now and then followed by paralysis of the soft palate, like that which is so often seen after diphtheria (see vol. i, p. 287). But I must confess that I should be rather disposed to think that in such cases the specific disease was really present, for marked swelling of the tonsil, and even suppuration in it, is by no means rare in diphtheria.

In the *treatment* of quinsy sucking of ice and the application of cold to the throat often gives more relief than anything else. But when an abscess is in process of formation the course of the disease may apparently be hastened by the use of fomentations and poultices externally and by steam inhalations. As soon as fluctuation can be felt an incision should be made with a guarded bistoury. Störk remarks that the best way of detecting a soft spot is to push the parts inward with one forefinger placed at the angle of the jaw, while the other forefinger in the mouth is carefully passed down over the inflamed structures from point to point. In opening a tonsillar abscess the cutting edge of the knife must be directed inwards and not outwards, lest the internal carotid artery should be wounded. Dr Mackenzie remarks that when the patient refuses surgical interference an emetic often leads to the immediate rupture of the abscess.

It many cases, however, it would seem that by suitable treatment quinsy may be made to abort, and the occurrence of suppuration be prevented when it would otherwise have taken place. Aconite has been recommended for this purpose, but according to many experienced practitioners guaiacum is even more efficacious. Dr Mackenzie administers it in the form of lozenges, each of which contains three grains of the resin; one of these, taken every two hours, seldom (he says) fails to arrest the disease at its first onset.

Chronic enlargement of the tonsils.—A not uncommon affection of the tonsils is a simple chronic overgrowth of their substance, or (as it is often called) a "chronic hypertrophy." They may be as large as chestnuts or even larger, and they are very firm and fleshy, smooth on the surface, but sometimes with cheesy or calcareous masses projecting from cavities in their interior. The affection may exist from early infancy, or develop itself during childhood or at puberty. In some cases it subsides as adult life is reached, and it seldom persists after the age of thirty. In children it is attended with many inconveniences; the mouth has to be kept open during sleep, and a snoring sound accompanies the breathing; some even say that a pigeon breast may result from the obstruction to the entrance of air into the lungs. There is something about the physiognomy of such patients that enables one to see at a glance what is the matter with them; Dr Mackenzie speaks of "the open mouth, the drooping eyelids, and the dull expression," and in addition to these peculiarities the voice is thick and nasal, and the act of deglutition (as in swallowing the secretions that accumulate in the mouth) is performed in a clumsy manner and with obvious effort. Another effect of chronic enlargement of the tonsils is deafness, which is attributed to a coincident swelling of the mucous membrane of the Eustachian tube. Dr Mackenzie says, too, that the senses of smell and of taste are often impaired; but sometimes the affection exists to a very marked extent without giving rise to any symptoms whatever.

The *treatment* may at first consist in the administration of cod-liver oil, iron, and other tonics, while the tonsils are every day brushed over with a solution of perchloride of iron, or smeared with powdered alum or tannin. But if there is much overgrowth of solid tissue, excision by means of the guillotine is almost always necessary. If hæmorrhage follows the operation, it is generally easily checked by making the patient suck ice; if not, Dr Mackenzie recommends that he should slowly sip half a teacupful of a strong solution of tannic and gallic acids, containing ʒvj of the former and ʒij of the latter to the ounce of water. The wounded parts remain sore for some days, and during this time the food has to be soft and bland, and the use of marshmallow lozenges is recommended as soothing.

Granular pharyngitis.—A very common affection of the back of the throat is one in which the pharyngeal surface appears dotted over with small prominences of about the size of millet-seeds, which may be either scattered, or closely packed together, or even confluent into ridges of varied breadth and length. At the same time the finer blood-vessels of the mucous membrane are seen to be dilated and tortuous. With regard to the nature of the granulations themselves there appears to be still room for further investigations. It is generally supposed that they consist of enlarged glands which have taken more than their share of a process of hypertrophy that likewise affects other structures, but not to so great an extent. Störk, however, says that the mucous membrane generally is in such cases often thinner than natural, and that what characterises the granulations microscopically is the absence of a superficial lining stratum of epithelium, large round swollen cells lying uncovered and exposed. As a rule, the surface of the throat is in such cases dry, there being apparently a deficiency of the normal secretions. But Dr Mackenzie describes an "exudative form" of the affection in which a viscid mucus is seen adhering in patches to the follicles, or in which their orifices are filled with a white material resembling cream cheese in appearance.

Dr Horace Green, of New York, was the first writer to give such an account of granular pharyngitis as fixed itself upon the attention of the profession; his names for it were "follicular disease" and "follicular inflammation" of the throat and air-passages. It is, indeed, by no means limited to those regions which are directly visible at the back of the mouth. Sometimes it spreads upwards towards the vault of the pharynx, sometimes downwards towards the larynx. Michel has recently insisted on the importance of thoroughly exploring with the laryngeal mirror every part of the fauces; for example, he says that when the rest of the surface is quite healthy, there may be a small patch of disease just behind one or both of the posterior arches of the palate; this may have been overlooked by one medical man after another, and yet it may really be the cause of a great deal of suffering to the patient, as is shown by touching it with a probe, and afterwards by the results of applying treatment to it. The subjective symptoms of granular pharyngitis vary widely in different cases. My own impression is that it very often causes no discomfort at all. Sometimes, however, it gives rise to a very troublesome feeling of stiffness or dryness in the throat, to a constant need for swallowing, to a pricking pain during deglutition (especially deglutition of the fluid secretions of the mouth), to a tickling sensation compelling a frequent cough that itself may be attended with soreness or pain, or to an incessant hawking, in the hope of getting rid of mucus, until actual retching may occur. In other instances, the chief complaints are associated with exertion of the voice. Speaking or singing may be attended with a sense of painful effort; the patient may be obliged to stop from time to time, in the middle of a sentence even, to swallow, or to clear the throat. Such cases have given to the disease the name of "clergyman's sorethroat;" it is indeed very common in those who earn their living by means of the voice, as in preachers and public singers, as well as in street hawkers and costermongers. As a rule, exposure to cold plays a conspicuous part in bringing out and in aggravating the symptoms of granular pharyngitis. Some writers also maintain that irritation of the fauces by excessive smoking or by alcoholic liquids should be mentioned among the exciting causes of the affection. An over-sensitive state of the nervous system appears in many cases to contribute largely towards increasing the severity of its subjective symptoms; some patients appear to be almost incapable of forgetting, even for an instant, the morbid feelings which they experience in the back of the throat; they are ever flying from one medical adviser to another, and they thoroughly deserve the name of hypochondriac which is freely given to them. Another element in the ætiology of granular pharyngitis appears to be an inherited predisposition. Dr Green speaks of three brothers, all clergymen, who were compelled by it to give up their official duties, and whose mother was also affected with it; and he also alludes to a large number of cases, recorded in his notes, of two or three members of a single family having been treated for it. The age at which it is most apt to occur is from twenty-five to thirty-five, and in men more often than in women. But Dr Mackenzie speaks of having seen it in children who were eight, six, or even only three years old.

In the *treatment* of granular pharyngitis the essential point appears to be the destruction of the granulations. So long as no local applications were used except gargles, inhalations, and brushing over the fauces with solutions of nitrate of silver, or even with the solid caustic, it was admitted by all candid observers to be an exceedingly intractable affection, and one that often ran on for years in spite of all that was done to cure it. In fact, as *gargling* with fluids is commonly practised, it seldom brings them into contact with any part of the fauces behind the anterior pillars of the palate. Guineir, of Cauterets, has clearly proved, in his brochure entitled 'Étude sur le Gargarisme Laryngien,' that when a person with his head thrown backwards makes fluid bubble about in the back of the mouth, while he goes on inspiring at regular intervals through the nose, the fluid is supported by the base of the tongue, the uvula, and the anterior pillars of the palate; any portion of it which passes further backwards is instantly swallowed. On the other hand, he has also shown that it is possible to teach patients to gargle in quite a different manner, and that the fluid then passes not only into the pharynx, but even into the larynx itself, resting directly upon the upper surface of the vocal cords. The directions are that the head should be slightly raised, the mouth but little opened, the lower jaw thrown forwards, so as to lift the chin. Having taken a small quantity of fluid into his mouth, the patient is to draw a deep breath through the nose, and then to allow the fluid to fall back into the fauces, while he endeavours to emit the sound of the double vowel *ê* (or the English *a* long). This, of course, means that the cords are brought together, and that the act of expiration is begun; while at the same time the epiglottis is raised so as to throw widely open the upper part of the laryngeal cavity. A bubbling sound is produced by the thin stream of air which passes outwards between the cords; this sound, Guineir says, is quite unlike that of ordinary gargling; it resembles rather the rattle in the throat made by persons who are moribund. The act of gargling within the larynx can be continued only so long as a slow expiration is being maintained. Before a fresh breath can be taken, the fluid must be thrown up into the pharynx, whence it often passes out through the nostrils. I have myself seen Guineir demonstrate with the laryngoscope the presence of a layer of fluid resting upon his own vocal cords. And he seems to have gained a considerable amount of success in the treatment of granular affections of the pharyngeal and laryngeal mucous membranes by making patients gargle in this way with the sulphur water of Cauterets.

Of late, however, far better results than had ever before been attained in this troublesome disease have followed the adoption of much more active measures. Dr Mackenzie applies a caustic paste to each granulation separately, touching on the same day only two or three, and sometimes only one, of them. It is easy to imagine that this practice must be tedious and must in many cases cover a long space of time. But in 1873 Dr Michel, of Cologne, drew attention in the 'Deutsche Ztschrft. f. Chirurgie' to the success which he had attained in about seventy cases by the use of the galvanic cautery. Since then many other observers have adopted this method. Its consists in applying a heated platinum loop to the granulations, so as just to destroy their surface; and, as a rule, the operation has to be repeated only three or four times, inasmuch as the effect is not limited to the part immediately cauterised, but extends to some distance around. There is little or no pain at the time, except when the pillars of the fauces are the parts touched by the instrument; the inflammation which follows can be kept within bounds by making the patient during the first few hours suck ice at intervals; there is nothing to prevent his continuing his usual avocations. Dr Foulis, of Glasgow, employed a small gas cautery for the same purpose; but this has the disadvantage that it must be heated before being passed into the mouth, whereas the platinum loop of the galvanic cautery is cold at the times of its introduction and of its withdrawal.

Adenoid vegetations in the vault of the pharynx.—In 1869 Dr Wm. Meyer, of Copenhagen, drew the attention of the Royal Medical and Chirurgical Society (vol. liii) to this affection, which he was almost the first to recognise, but which has proved to be exceedingly common. It consists in the presence of masses of various shapes and sizes, growing most frequently from the posterior wall or from the roof of the pharynx, but sometimes also from the sides of that cavity, or even from the upper surface of the soft palate, never from the back of the nasal septum. They are described as occurring in three forms, the *cristate*, the *cylindrical*, and the *flat*. They are sometimes soft, and sometimes hard. They contain many vessels, especially veins; and they are otherwise made up of a scanty areolar network, having its meshes filled with lymph-cells. Their epithelium may either be ciliated, or of pavement form, according to their exact seat. Their colour is generally the same as that of the more or less congested mucous membrane in their vicinity, but they may have a slightly yellowish hue.

It is often possible to tell by the way in which a person speaks, and even by the expression of his face, that he is affected with pharyngeal vegetations. The peculiarity of the speech consists partly in an inability to utter the sounds *m*, *n*, *ng*, so that instead of "common" the patient says "*cobbod*," instead of "nose" "*doze*" or "*loze*," instead of "song" "*sogg*;" partly in a loss of the resonance naturally given to the voice in the nasal cavities. Meyer characterises it as a "dead" mode of utterance.* The peculiarity of the facial expression depends upon the obstruction to the passage of air through the nostrils in breathing. This causes them to appear narrow and collapsed from disease, and the nose itself thinned and flattened from side to side. It also compels the patient to keep the mouth more or less constantly open; and since the orbicularis oris no longer gives support to the other muscles of the face, the countenance acquires a vacant stupid aspect, often increased still further by an odd trick of twisting and pouting the lips. Other symptoms are a feeling of fulness in the upper part of the fauces, as though there were a foreign body there, a secretion of thick greyish or greenish mucus which glides down the pharynx and compels the patient to be constantly clearing the throat, the presence of blood in the mouth, especially on first waking in the morning, and a more or less habitual headache. But what most commonly causes patients who have vegetations in the vault of the pharynx to seek medical advice is the impaired state of their hearing. Sometimes there is only occasional deafness with a tinnitus, especially if they happen to take cold, but in many cases the aural symptoms are far more serious and lasting; there is chronic catarrh of the tympanic cavity, and the drum may even become perforated, giving exit to a purulent discharge. It is to be observed, too, that various affections of the fauces are apt to be associated with the presence of pharyngeal adenoid growths. The tonsils may be enlarged, there may be granular pharyngitis, the uvula and the soft palate may be thickened. There may also be catarrh of the anterior parts of the nasal cavities, though in the majority of cases the secretion of the Schneiderian membrane is rather deficient than excessive.

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* [It depends upon the occlusion of the posterior nares preventing the nasal cavity from acting as a resonator. Hence the explosive vocal sounds take the place of the continuous vocal or "nasal" sounds. The same effect is produced by closing the anterior nares.—Ed.]

The easiest method of detecting with certainty the presence of vegetations in the vault of the pharynx is to explore the upper part of the pharynx with the forefinger. This is to be passed between the tongue and the roof of the mouth, and insinuated by the side of the uvula until it glides upwards behind the velum. It is then carried along the posterior edge of the septum of the nose, and turned in various directions until every part of the space has been thoroughly examined. If necessary a probe introduced through the nostril may be used to bring the individual vegetations in contact with the finger. The examination may cause some nausea, and may even be followed by pain in the back of the head; there is often a good deal of bleeding from the growths when they are touched. Rhinoscopy is seldom of much assistance in diagnosis; the cases in which vegetations can be most satisfactorily seen are those of persons with a cleft palate, an affection that appears to be rather frequently associated with their presence.

The systematic examination of children at schools in Denmark, England, and Holland has shown that from 1 to 5 per cent. of them have been affected with pharyngeal adenoid growths. The disease is said to be more frequent when the climate is cool and damp. It is seen chiefly in persons under the age of twenty-five years, and it appears often to be congenital or to date from very early childhood. It often affects several members of the same family; Meyer thinks that it is more common in boys than in girls. It is said to be often a sequela of measles, and also to be traceable in many cases to "colds." Its relation to scrofula is doubtful; according to Wiesener, of Bergen, it may lead to an infiltration of the cervical glands such as is commonly called scrofulous.

When adult life is reached, adenoid vegetations in the vault of the pharynx appear commonly to shrink and to disappear spontaneously. Nevertheless it is important to remove them as soon as their presence is detected, especially on account of the damage which they do to the organs of hearing. Sometimes cauterisation with solid nitrate of silver suffices to destroy them, but they generally have to be scraped off by suitable instruments, of which descriptions may be found in Meyer's several papers, and especially in the 'Transactions of the International Congress of 1881.' The chief of them consists in a small oval ring, with a sharp though not absolutely cutting edge mounted on a slender stem. This is passed backwards through the patient's nostril, and is guided to the bases of the vegetations by the operator's left forefinger introduced through the mouth. The operation causes little pain but profuse hæmorrhage, easily checked by the injection of cold water containing salt and carbolic acid. It is often followed by headache, and even by slight stupor for a few hours. It sometimes has to be repeated once or oftener, because all the vegetations are not completely got rid of on the first occasion. If any remains of them are left they are sure to grow again. Meyer therefore insists greatly upon the importance of an "after-treatment" consisting in cauterisations with nitrate of silver and daily injections of a solution of bicarbonate of soda, or of chlorate of potass. So long as there is any soft tissue to be felt which bleeds when touched these measures should not be discontinued. The effect of the complete removal of pharyngeal adenoid growths is quickly to restore the natural speech, to change in a surprising maner the expression of the patient's face, and in many cases to bring back the sense of hearing.

DISEASES OF THE ŒSOPHAGUS.*—This part of the alimentary canal is remarkably free from the slighter inflammatory affections which are so common both above and below it. Its thick layer of squamous epithelium seems to protect it from all but the most violent irritation, and it has neither the rich blood-supply nor the active secreting functions, nor the abundant lymphatic tissue, which elsewhere in the alimentary tract become the sources of disease.

Chronic *inflammation* of the gullet with thickening of the mucous membrane is, however, seen either as the result of external pressure, such, *e.g.* from a thoracic tumour or an aneurysm, or in a more diffuse form in chronic valvular disease of the heart. In the former case the thickened and opaque mucous membrane is, in external appearance as in pathology, like the "corns" produced by friction on the pericardium, or the white patches of the tongue where it is touched by a tooth or other mechanical irritant. In the latter case extreme venous congestion may be discovered after death, with desquamation of the upper layers of epithelium, a condition comparable to the far more important congestive catarrh of the stomach in similar disease of the heart.

Occasionally, when no source of pressure or irritation can be ascertained, the mucous membrane is found (at times over a considerable space) to be covered with minute *papillæ* which may be large and circumscribed enough to deserve the name of a papilloma.

Functional stricture.—The above pathological conditions are without clinical significance; there are, on the other hand, functional affections of the gullet which are at present without an anatomical explanation. Of these the most important is what has been called "Spasmodic Stricture." The patient is usually young, most often a girl at the age between puberty and childbearing, when functional neuroses are most common. It also, however, occurs in male subjects, and one of the most marked and obstinate cases I have seen was in a boy of fourteen. In most cases the neurotic or "hysterical" character of the affection is sufficiently evident, and the easy passage of an œsophageal sound completes the evidence.

Another condition which is probably at first functional is *regurgitation* of food. This differs from the gastric regurgitation, or as it may be termed "rumination," which will be described under disorders of the stomach (*infra*, p. 128); for here the food never reaches the gastric cavity, but is detained in the gullet. Œsophageal regurgitation appears to begin rather as a bad habit than a disease; but whether or no there be any structural lesion as its original cause, there is frequently, or perhaps always in confirmed cases, a pouch formed in the lower part of the gullet, in which food collects before its regurgitation. Such a *dilatation* is said to be more frequent in men than in women. It usually involves the whole thickness of the œsophagus, but cases have been described in which the mucous coat alone has protruded between the muscular fibres. A classical case of this curious condition was published in the thirtieth volume of the 'Medico-Chirurgical Transactions' (1849) by Mr Worthington, of Lowestoft.

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* [This section, pp. 109—112, not begun by Dr Fagge, was supplied by the Editor.]

An œsophageal pouch is most frequently met with, not as a primary lesion, but as the result of a stricture immediately below it. It has occasionally proved to be the result of a mere narrowing of the gullet at its cardiac end. Such a case is described and figured by the author in the 'Guy's Hospital Reports' (3rd series, vol. xvii, p. 414) where at last a cancerous growth developed and proved fatal at eighty-four, forty years after the appearance of dysphagia. In the same paper is figured a dilated œsophagus resulting from a simple, non-traumatic stricture of the cardia, which was described by Dr Wilks in the seventeenth volume of the Pathological 'Transactions' (p. 138). Regurgitation and dysphagia had existed all the life of the patient, a healthy farmer, who had once consulted Sir Astley Cooper for œsophageal stricture, and who died at seventy-four of acute pneumonia. Mr Durham has discovered two cases of dysphagia and simple stricture, recorded by Sir Everard Home ('Practical Observations on the Treatment of Stricture in the Urethra and in the Œsophagus,' 1821, vol. ii, p. 398). One of these showed, post mortem, a fold of mucous membrane, which narrowed the gullet just opposite the cricoid cartilage.

So-called *dysphagia lusoria* demands a word of notice. The term was first applied by Dr Bayford, of Lewes, to a case (probably of spasmodic stricture of the gullet) in which the right subclavian artery arose from the third part of the aorta, and passed to its distribution between the œsophagus and trachea. This *lusus naturæ* was probably a mere coincidence. The much more frequent abnormality of the right subclavian arising from the third part of the arch, and passing *behind* the trachea and œsophagus, between the latter and the vertebræ, is found, post mortem, in persons who have never experienced difficulty in swallowing.

Organic stricture of the œsophagus are clinically divided into the simple or non-malignant, and the cancerous.

Simple stricture can in most cases be traced to a *traumatic* origin, most frequently to irritant poisons, such as the mineral acids. Constriction of the gullet by external cicatrices or pressure of diseased vertebræ, abscesses, aneurysms, cancerous mediastinal glands, or possibly a distended pericardium, will of course have the same results as true stricture or contraction of its walls, and will only be distinguishable during life by evidence of the presence of the external compressing cause.

Stricture is occasionally due to contraction of a *simple ulcer* of the œsophagus; which is, however, a very rare affection compared with the corresponding lesion in the stomach, or even the duodenum. Above a stricture there may frequently be seen hypertrophy, with or without dilatation of the muscular walls of the gullet. There is no doubt that *syphilitic* ulcers, probably of a tertiary gummatous nature, may give rise to contraction of the œsophagus. ('Guy's Hospital Reports,' vol. xvii, ser. 3, 1872, p. 413.)

Malignant stricture of the œsophagus is always primary and is usually of the epithelial, keratoid, or flat-celled variety (cf. vol. i, p. 101). Adenoma or glandular cancer has occasionally been observed, but true examples of the encephaloid or scirrhous variety are most rare.

The most frequent position is often stated to be in the middle of the tube, opposite the bifurcation of the trachea. Less in frequency is the upper portion, where cancer spreads so as to be described indifferently as pharyngeal or œsophageal. Lastly, a malignant stricture is occasionally met with at the entrance into the cardiac orifice of the stomach, and it is here that its presence is most apt to be overlooked, the diseased portion being left in the diaphragm when the stomach and œsophagus have both been removed, as Virchow long ago observed. The foregoing statement is that of Wilks, Rindfleisch, and Klebs. Many text-books, however, follow Rokitansky's original assertion that the upper part is the most frequent seat and the lowest the rarest. Förster, Moxon, Payne, and Coats say that the commonest seat of cancer of the œsophagus is its lowest third; and this statement is confirmed by the careful analyses of Petri and Zenker. In 58 cases collected by these two writers 4 were in the upper, 14 in the middle, and 24 in the lower third. The remaining 16 cases occupied the middle and adjacent parts also. The latter group of cases is the disturbing element which has probably affected the classification of statistics. It still, however, remains true that opposite the bifurcation of the trachea is a frequent seat of œsophageal cancer, and that cancer of the extreme cardiac end is rare. In 13 cases collected by Dr Moore, of St Bartholomew's Hospital, the lower third was affected in 7, the middle third in 5.

The malignant growth is sometimes a mere cartilaginous ring like an annular stricture of the colon, but more frequently it forms an ulcer which only partially encircles the tube and infiltrates and spreads up and down for an inch or even more. Dr Hughes Bennett records a rare case of double cancerous stricture ('Prin. and Pr. of Med.,' p. 453).

The progress of epithelioma of the gullet is slow, and it rarely affects more than the neighbouring lymph-glands of the mediastinum. Beside causing death by inanition, it may produce fatal hæmorrhage, or, again, may perforate by sloughing, involve the vagus nerves, or open anteriorly into the trachea or lung. Death often occurs from pneumonia or pleurisy. In one case under the care of the Editor the primary stricture was latent, and the first symptoms were pain and afterwards paraplegia caused by secondary cancer of the vertebræ.

Myomata, polypi, besides the warty growths above mentioned, and other non-malignant growths, have occasionally been observed in the œsophagus.

Malignant stricture is more common in men than in women, and is rare before middle life.

Course and diagnosis.—Its early symptoms are slight and its progress insidious. Difficulty in swallowing solid food is commonly the first complaint. Pain, though occasionally severe, is often long before it appears, and is sometimes almost absent. Gradually the patient finds it more and more difficult to swallow soft food, and at last even liquids, and increasing emaciation is the result. So latent, however, are the symptoms, that the disease has sometimes been only discovered after death, though a tradition of a diagnosis once made by Sir Astley Cooper shows how the aspect and age of a person suffering from this disease may be recognised by experienced observation or by a shrewd guess. The cautious passage of a bougie is the only decisive proof of the nature of the disease, and also gives a criterion of its position and of the calibre of the tube. Hamburger's methods of diagnosis by auscultation deserve mention (Erlangen, 1871: see also Dr Albutt's paper. 'Brit. Med. Jour.,' ii, 1875). Of less practical importance is the ingenious attempt to obtain a view of the gullet by Waldenburg: his instrument is figured in the 'Berlin klin. Wochenschrift,' 1870, p. 580.

The *treatment* of stricture of the œsophagus is purely mechanical. When free from ulceration, the stricture may be benefited by the frequent passage of a bougie, or by passing a tube into it and leaving it in the stricture for hours, or even days, so as to effect a dilatation. Even in cancerous strictures, so long as there is no ulceration, the occasional passage of an olive-shaped bougie frequently affords great relief, but the utmost care and gentleness is essential, or fatal perforation may ensue. As Mr Bryant well puts it, when a patient complains of difficulty in passing food onwards down the gullet after the act of swallowing, and of its return into the mouth, the practitioner should first think of thoracic aneurysm, then of cancer, and then of some other kind of ulceration. The dread of such a catastrophe as perforating an aneurysmal sac, or thrusting a bougie into the pleural cavity, will, however, be the best safeguard against its occurrence. No force which an intelligent hand could use will perforate an intact mucous membrane. When perforation, or even ulceration, has already taken place, no one would willingly risk the passage of a tube. But where there is no evidence of ulceration the practice is defensible and beneficial. The *sonde œsophagienne à demeure*, above mentioned, was advocated by Mons. Krishaber, at the International Medical Congress of 1881 ('Transactions,' vol. ii, p. 392). He advises its passage through one of the nostrils rather than the mouth, and in proof of the tolerance of the instrument relates four cases in which the tube remained continuously *in sitû* for 46, 126, 167, and 305 days respectively, Feeding, of course, takes place entirely through the hollow œsophageal sound. I have seen a similar plan carried out by Mr Symonds at Guy's Hospital with good success.

When obstruction has become complete, life may be preserved for a time by nutrient enemata. For this purpose small quantities of peptonised food without salt or alcohol are best employed ; and in many cases raw eggs, beef tea, and pancreatised milk, are well retained and absorbed. But often the rectum rejects the nutriment; after a time this result is almost sure to occur, and even in the most favourable cases the patient is insufficiently nourished. In acute cases of disease, or of injury or operation about the mouth and throat, or even while a gastric ulcer is given time and rest to heal, the treatment by rectal alimentation is most valuable. But when, as in stricture of œsophagus, the disease is progressive, it is far better, so soon as occlusion occurs, and no liquids even can reach the stomach, for the operation of opening the stomach to be faced before the patient's strength and endurance have been exhausted.

The operation of gastrotomy, or *gastrostomy*,* was first performed by Sédillot, in 1849, and was introduced into England by Mr Cooper Forster. It has since been amply justified by the long periods of life and comfort which it has afforded to patients who would otherwise have died by one of the most painful deaths—that from thirst. In a case brought before the International Congress in 1881 ('Trans.,' p. 456) by Mr R. J. Pye-Smith, of Sheffield, the patient survived a year and a half, and in a second case (for malignant stricture) by the same surgeon life was prolonged for 128 days. For similar cases see a paper by Dr Gross, jun., in the 'American Journal of Med. Sc.' for 1884.

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* Gastro-stomie was the French surgeon's original term. Mr Bryant follows Dr Pooley, of New York, in defining "gastrotomy" as opening the stomach for removal of a foreign body.

DISEASES OF THE STOMACH

Acute Dyspepsia.—*Acute catarrhal gastritis—Its symptoms, causes, histology, and treatment—Acute paralytic distension—Acute suppurative gastritis.*

Chronic Dyspepsia.—*Atonic and chronic catarrhal forms—Symptoms, causes, anatomy, diagnosis, and treatment.*

Functional Disorders.—*Gastralgia—Anorexia, &c.—Eructation and Pyrosis—Vomiting, &c.—Hæmatemesis.*

Gastric Ulcer.—*Anatomy—Pathology—Ætiology—Symptoms—Event and duration—Treatment.*

Cancer of the Stomach.—*Carcinoma of the pylorus and of the body of the stomach—Sarcoma—Colloid cancer—Symptoms—The tumour—Consequent dilatation—Gastro-colic fistula—Duration—Diagnosis—Treatment—Fibroid induration of the stomach—Concretions.*

In health, as everyone knows (or has known) by experience, the process of digestion is unattended with any kind of sensation; we ought not to be conscious that we have stomachs. But under morbid conditions, it may be accompanied with unpleasant feelings, varying in degree from a slight sense of weight or discomfort, up to agonizing pain. These may be symptoms of severe disease of the stomach, or of some slight organic change, or of a mere functional derangement. Many of the less serious disorders of the stomach are commonly grouped together as Dyspepsia, or indigestion.

1. Acute Dyspepsia.—It is not uncommon in persons who were before perfectly well, for the stomach to resent some particular meal, on account either of its quality or its quantity. Such instances must have come within the knowledge of every medical man. Those which I remember best are as follows:—A schoolboy eats hastily a large quantity of grapes, skins and all; in a few hours he feels ill, and presently rejects the contents of his stomach; next morning he is well. A man, in the month of July, has the usual order of his meals disturbed by social engagements; he partakes twice in the same day of a variety of dishes, and probably eats much more than he really requires; he goes to bed with an uneasy feeling at the epigastrium, and in the night he vomits in surprising quantity the food that he had swallowed, almost unchanged, but dry, without the liquid with which he had washed it down; his discomfort is then at an end, and he falls asleep. Each of these patients brings up a little bile when his stomach has emptied itself of its accumulated contents. Formerly this fact would have been regarded as a sufficient ground for calling the complaint a "bilious" attack. But we now know that, in consequence of the antiperistaltic movements induced in the duodenum, bile enters the stomach whenever there is much vomiting, and is of course rejected in its turn.

For attacks such as I have described the name of acute indigestion is fairly applicable. But it is usual to describe under that name a somewhat different class of cases, which are of longer duration. And since these are believed to depend upon a catarrhal inflammation of the gastric mucous membrane, the terms "acute indigestion" and "acute catarrh of the stomach" are commonly regarded as synonymous.

Acute gastric catarrh.—In different cases, the symptoms vary somewhat. In addition to a sense of weight and oppression at the epigastrium, which is common to all forms of dyspepsia, there is almost always actual pain. This is usually a dull aching, but sometimes it assumes a burning, stabbing, or griping character. One patient who came to me complained chiefly of a soreness at the left side of the sternum and about the left scapula; another felt more pain in the back than in the abdomen. In some cases, however, the epigastrium is tender, and it often feels full and tense. The patient is thirsty and eager for cold and acid drinks; but he has generally no appetite, and often experiences a loathing for all kinds of food. One man told me that his appetite was good, but that he felt afraid to eat anything, knowing what discomfort it would cause him. The breath is offensive. The tongue is often surprisingly foul and coated with a thick yellowish white fur; this is one of the most characteristic indications of the disease. Nausea and retching are generally prominent symptoms, but one patient whom I saw had only a slight tendency to retch, and complained rather of the sour gases which came into his mouth by eructation. Sometimes ineffectual efforts at vomiting are made, but more often partially digested food, with a very acid taste, is rejected; or a quantity of whitish mucus. Aching in the back and limbs, malaise, and depression of spirits are also complained of. There may be more or less fever, but the skin is generally moist. In one of my cases the attack began with shivering. A herpetic eruption sometimes breaks out upon the lips and chin.

An attack of this kind may last from a day or two to a week, or even longer; if injudiciously treated it may pass into chronic gastric catarrh. Prolonged cases have been described by French writers as "*Embarras gastrique.*"

Various *causes* of acute gastric catarrh are mentioned by writers. The cases most readily accounted for are those in which it follows the ingestion of some irritating substance. I attended one patient in whom a severe and protracted attack came on the day after he had dined on venison and champagne. In infants improper food frequently causes attacks of this kind, which are commonly, but not always, complicated with diarrhœa. Prostration is a very marked feature in such cases, and death often results. Decomposing meat or vegetables, and shell-fish under certain conditions, are especially apt to give rise to acute catarrh of the stomach. It may be the effects of the inhalation of dust or vapour given off from wall papers containing arsenic. Dr Wilson Fox mentions the case of a healthy child who, after sleeping in a room lined with such a paper, was seized with severe vomiting, and even brought up blood. I do not mention the direct ingestion of irritants like arsenic and antimony, which cause violent inflammation of the stomach, as described in works on toxicology: for this there is no analogy in the effects of ordinary disease.

Exposure to changes of temperature is said to be another cause. Dr Wilson Fox quotes from Guipon the case of a workman, who on several occasions was seized with vomiting and pain in the stomach after being exposed to the heat of a furnace. And he says that attacks of this kind are especially apt to occur in changeable weather, as in the spring and later autumn, when cold and high winds prevail, as well as during the severe heats of summer and early autumn. Epidemic influences are also mentioned by writers. Dr Fox cites Chomel as having observed that gastric catarrh was frequent where cholera was raging; and Dr Fox himself noticed the same thing in 1866. Lastly, it is said to be a frequent complication of scarlatina, erysipelas, measles, diphtheria, smallpox, puerperal fever, &c. And at any rate every medical man is aware that each of these diseases is apt to be ushered in by repeated acts of vomiting, which, however, last only till the characteristic rash makes its appearance.

As acute gastric catarrh has no tendency of itself to destroy life, one might fairly expect that its *morbid anatomy* would be a *terra incognita*. But, half a century ago, a remarkable case occurred, in which an American named Alexis St Martin, after receiving a musket-shot wound in the left side, acquired a fistulous opening into his stomach, so that part of its mucous membrane was permanently exposed to view. The case was carefully investigated by Dr Beaumont, and he relates that deep red pimples sometimes appeared, which afterwards became filled with purulent matter; and at other times irregular circumscribed red patches, small aphthous crusts, and abrasions of the lining membrane, leaving the papillæ bare. These diseased appearances, when considerable, were attended with dryness of the mouth, furring of the tongue, thirst, and acceleration of the pulse, and the secretion of gastric juice was suspended, so that food remained undigested for twenty-four or forty-eight hours, or more; although liquids were absorbed as soon as they were swallowed. Mucus was also poured out by the surface of the stomach, and slight hæmorrhages sometimes occurred. Now, it is true that symptoms were by no means constantly present where the mucous membrane presented these appearances, but the case is nevertheless one of the utmost value, as showing that the stomach is susceptible of morbid changes which, if they could generally be seen, would arrest attention. For, in the post-mortem room, such changes can seldom be observed satisfactorily. After death, the lining membrane of the stomach is commonly acted upon by its contents, so that it becomes softened and pulpy, or may be entirely dissolved over a more or less extensive area. All the coats of the organ, indeed, may in this way be perforated, a large ragged aperture being left. As might be expected, this is generally at the back of the stomach, on which the contained matters rest while the body lies with its face upwards. Moreover, in the stomach, as in all other parts, congestion is very apt to subside after death, so that it can no longer be detected. We have, however, seen that in obstructive diseases of the heart the gastric mucous membrane is found intensely reddened and ecchymosed and lined with mucus; and great reddening is also seen in the bodies of those accustomed to drink spirits to excess. But it is probable that the changes which Dr Beaumont described would no longer be visible in a post-mortem examination. Writers, however, describe certain other appearances as characteristic of acute gastric catarrh. Dr Wilson Fox mentions an increased opacity of the mucous membrane, with swelling and diminution of its consistence. Under the microscope, he says, the secreting cells and the nuclei are swollen, irregularly distending the tubules. They are filled with granular matter, and in severe cases they often break down into a granular *débris*. Dr Cayley and Dr Fenwick have found in fluids from the stomach tube-casts comparable with those which are well known to occur in the urine in Bright's disease. A drawing of these is to be seen in the 47th volume of the 'Med.-Chir. Transactions.'

In addition to these changes, Dr Wilson Fox describes an increase in the size of the solitary follicles of the stomach, which appear as small white specks, scattered over its surface, and which sometimes ulcerate, forming little cup-shaped depressions. The interstitial tissue between the tubules also becomes infiltrated with leucocytes.

Now, it is important to note that the cases in which these appearances have been found have not been simply cases of gastric catarrh; for these do not terminate fatally. The observations of Dr Wilson Fox and others have been made in the bodies of those who had died of scarlatina, diphtheria, pneumonia, or some other disease. They undoubtedly prove that the diseases in question are attended with morbid changes in the stomach, but not, as it seems to me, that these diseases are to be regarded as so many causes of acute gastric catarrh, in the clinical sense of that term. For, although in a case of this kind the patient may be more or less sick and have a furred tongue, yet it can hardly be said that distinctive symptoms of catarrh of the stomach are present, or that the gastric affection modifies in any way the course of the disease.

The *diagnosis* of acute gastric catarrh is generally easy. Both Bamberger and Dr Wilson Fox observe that enteric fever in its early stage is the disease which is most likely to be confounded with it. According to Dr Fox it is common at all ages, but I cannot say that it has been so in my experience. I have seen very few well-marked instances of it in adults, nor do I remember any case which could have been mistaken for one of fever.

In the *treatment* of an attack of acute indigestion the first thing to be attended to is the diet. In mild cases, Dr Fox says that complete abstinence from food for twenty-four hours often does most towards effecting a cure. In severe and protracted cases nutrient enemata may often be used with much advantage. If any nourishment is given by the mouth it should be milk in very small quantities, and diluted with soda-water or lime-water. Persons who cannot take milk must have veal or chicken broth, or beef tea. As the symptoms subside, light farinaceous puddings may be allowed, but no solid meat, until they have entirely passed off. Unless there be great prostration the patient should abstain altogether from alcoholic liquids; champagne is not so suitable in this as in other forms of irritability of the stomach. If a stimulant appears absolutely necessary brandy well diluted is the best.

In infants at the breast the quality of the milk must be carefully inquired into. Dr Fox mentions the occurrence of menstruation in the nurse as a reason for taking the baby from her. Sometimes it is advisable to keep it from the breast for a few hours, giving it only a little rice-water or very diluted milk at intervals. In infants brought up by hand, the substitution of asses' milk for cows' milk is sometimes effectual; but too often one can save the child only by promptly engaging a wet nurse.

Fomentations or poultices may be applied with advantage, and in adults, if the pain is continuous, Dr Fox recommends leeches.

But the most important questions concern the administration of emetics and purgatives. Dr Fox says that an emetic may be administered "when the presence of undigested food is indicated by cramp-like pain, nausea, ineffectual attempts to vomit, and faintness." Two cases of Sir Thomas Watson's may here be alluded to. One was that of a person who had been taking large quantities of cream with his tea and coffee. After suffering for several days under severe gastric pain and disorder, he threw up a mass of hard curd like a small cream cheese, and he was at once completely relieved. In the other case a similar fit of indigestion terminated in the ejection of a mass of snuff. It is certain, therefore, that irritating matters may remain for a considerable time in the stomach. But, on the other hand, as Bamberger remarks, one must not trust too much to the circumstance that the patient continues to experience uneasy sensations, for these may continue long after their cause is removed, just as one goes on imagining that there is a foreign body in the eye long after it has been got rid of. Bamberger, indeed, gives a caution against the use of emetics or purgatives, which, he says, have caused catarrh of stomach much more often than they have cured it. Dr Wilson Fox, too, says that antimony and even mustard are to be avoided, and that ipecacuanha with large draughts of lukewarm water or of infusion of chamomile is the best emetic in these cases. He recommends, however, rather active purgatives; from three to eight grains of calomel followed by a black draught, or by castor-oil; or a dose of blue pill and compound colocinth pill, with a seidlitz powder afterwards. Dr Beaumont certainly found in the case of Alexis St Martin, already referred to, that the administration of calomel (which he introduced into the stomach in rather large doses) was followed by a subsidence of morbid appearances in the gastric mucous membrane. But I am nevertheless inclined to agree with Bamberger in thinking that we ought to be very careful in prescribing purgatives in cases of supposed acute gastric catarrh. I can never forget a case which I diagnosed as of this nature, and which proved to be one of acute suppurative peritonitis. A bank clerk felt poorly one day after having eaten some pears in the afternoon. In the night he woke up with epigastric pain and vomiting. A medical man was not sent for for two days, and when he came he gave a mild aperient. This operated, and a day or two later the sickness subsided. There was a little delirium about the third day. The pulse was at no period of the case over 100; the temperature ranged from 100° to 101°. I was asked to see him on the sixth day. He then appeared to be better, the sickness and pain had ceased, he had begun to take food again. The pulse was about 90, of fair volume; the temperature exactly 100°. Except that the countenance was sunken, and that the eyes were surrounded by deep brown rings, there appeared no reason for alarm, and I concluded that the attack had been one of acute indigestion, and that the patient was in a fair way to recover. Within twenty-four hours, however, he died, and it turned out that there was diffused peritonitis, set up by ulceration of the vermiform appendix.

But whatever may be said as to the propriety of beginning the treatment of a case of this kind with an emetic or with a purge, all writers are agreed that when once given it should not be repeated. Sedatives and antacids are the principal remedies. Sucking small pieces of ice often gives much relief. When there is severe vomiting, Dr Fox recommends opium, but I have no doubt he is right in saying that in milder cases it may do harm rather than good. For such cases he speaks of bismuth as being particularly useful; and this accords with my experience. Ten grains of the subnitrate with as much carbonate of soda, and a little morphia or dilute hydrocyanic acid, will often give immediate relief. Vichy or seltzer water may be prescribed with much advantage, a pint or a pint and a half being given daily. Effervescing medicines are also useful. But the patient should not be allowed to take liquids in such quantities as to distend the stomach. During convalescence the remedies that will hereafter be recommended for chronic dyspepsia become applicable. But Dr Fox gives a caution against the use of the bitter tonics in protracted cases of acute catarrh of the stomach; "they often," he says, "perpetuate a chronic inflammatory action."

Acute gastric dilatation.—I have implied, that in adults acute catarrh of the stomach is itself unattended with danger. But it may be a question whether certain very rare cases, in which rapidly fatal collapse occurs after gastric symptoms, of at first no particular severity, have lasted a few days or a week or two, ought not to be regarded as due to the supervention of a further morbid state upon one of catarrhal inflammation. I have myself seen two instances of the kind. These I described in the 'Guy's Hospital Reports' for 1872-73, under the name of "acute dilatation of the stomach," but a better title would perhaps be "acute paralytic distension." One case occurred in a man, aged thirty, who had for some time been in the hospital under Dr Owen Rees and was supposed to have incipient phthisis. He was seized with persistent vomiting; he passed no urine; and he gradually became collapsed. On examining him on the third day I found that the abdomen was retracted and that its walls were rigid. There was dulness above the pubes and half way up to the umbilicus. This might have been attributed to distension of the bladder, but a catheter had been passed and no urine could be obtained. Moreover, a splashing sound was obtained by manipulation of this region or of the iliac fossæ. He died the same afternoon. On post-mortem examination the stomach was found to fill the whole abdomen and to contain a large quantity of fluid. But when removed from the body it shrank back to about its natural size, showing only a number of fine white striæ on its serous surface, apparently analogous to "lineæ gravidarum." The other case I saw in consultation with Mr Hooper, of Bermondsey. A man, aged twenty, had for a fortnight been suffering from abdominal pains and repeated vomiting. Two days before my visit the sickness ceased entirely, but he nevertheless became worse in all other respects. I found him with a sunken countenance, his eyes glassy and surrounded by deep rings of pigment, his breath nauseously sweet. His abdomen was generally distended, but the right hypochondrium was flat, and passing downwards and to the right above the navel a line could be seen which I recognised as indicating the upper border of the stomach. On manipulation of the lower part of the abdomen, I detected fluctuation and obtained a splashing sound. I therefore diagnosed dilatation of the stomach, and as soon as I could procure a stomach-pump, I had a long tube passed down the œsophagus. A greenish fluid was ejected through it and by its side with considerable force. The pump was then connected with it and no less than *seven pints* of the same fluid were removed by its means. The abdomen became deeply hollow while this was being done. The patient said that he felt much relieved, but he died four hours afterwards. The post-mortem examination showed, as I expected would be the case, that the stomach had returned to its natural size and form; but there was a sloughing abscess behind the duodenum, communicating with the bowel. This must doubtless have caused the patient's death under any circumstances. But I think it may fairly be hoped that in an uncomplicated case like the former one the prompt use of the stomach-pump would afford a chance of saving the patient's life if an early diagnosis could be made. It seems clear that the cessation of vomiting is due to a paralytic state of the gastric muscular coat, comparable with that which occurs in the bladder in cases of retention of urine. I do not know what is the origin of the large quantity of fluid which the stomach contains in these cases. Unless it is merely what has been swallowed, it must indicate an irritated or inflamed condition of the lining of the organ. I regret that the mucous membrane was not examined microscopically in either case.

The physical characters which indicate acute paralytic distension of the stomach during life are: 1. A rapidly increasing distension of the abdomen, which is unsymmetrical, the left hypochondrium being full while the right is comparatively flattened. 2. The presence of a surface marking which descends obliquely from the left hypochondrium towards the umbilicus and which corresponds with the lesser curvature of the stomach. This seems to move up and down each time the patient breathes. 3. Dulness and fluctuation in the pubic region with resonance over the front of the abdomen. 4. The production of a splashing sound on manipulation. It is to be observed, however, that in one of my cases the first two of these signs were absent.

Phlegmonous gastritis.—Another rapidly fatal disease of the stomach is acute diffused suppurative inflammation: a still more rare affection than paralytic distension. Bamberger mentions it in Virchow's series of handbooks (1855), but Rokitansky had (p. 260 of his vol.) previously described it as suppurative inflammation of the submucous connective tissue, and refers to older cases recorded by Monro, Lieutaud, and Albers. Ackermann collected thirty cases, mostly puerperal. It has been compared to phlegmonous erysipelas or "pseudo-erysipelas" of German pathologists. Wilks and Moxon mention hepatic abscess as a result. A typical case of this rare disease is recorded by the author in the 'Path. Trans.,' vol. xxvi, p. 81.

Chronic Dyspepsia.—The most common and the most important of the less dangerous disorders of the stomach are those which are chronic in their course; and these are usually referred to when indigestion is spoken of. Writers on gastric affections describe two forms of chronic dyspepsia; one they call "atonic," the other "chronic gastric catarrh," or "chronic inflammatory dyspepsia." It is admitted, however, that there is great difficulty in distinguishing between them. And the most convenient course will be for me first to give the clinical history of the former, and then to contrast with it that of the latter affection.

A person who has *atonic dyspepsia* complains of a sense of weight and uneasiness after food, which may last for some hours, or even up to the next meal. The seat of these unpleasant sensations is usually the upper part of the abdomen; but sometimes, according to Dr Wilson Fox, they are referred to behind the sternum, so that a feeling of dyspnœa is experienced, or an impression that the œsophagus contains some foreign body. There is rarely actual pain, unless it be as the result of flatulence. There is no tenderness of the abdomen, pressure rather gives relief. Eructations of gas, and even of undigested food, are not uncommon; and these often cause an offensive or rancid taste, which is attributed to the formation of butyric acid. The appetite is generally deficient, there may be a distaste for food of all kinds even though the want of it gives rise to a sense of exhaustion. Thirst is generally absent, indeed the ingestion of fluids often seems to aggravate the symptoms. The tongue is broad, pale, and flabby, it is marked at its edges by the teeth, but is not generally thickly furred. Constipation is always present. According to Trousseau this is due to the close sympathy between the stomach and the intestine, of which sympathy he gives an odd illustration in the fact that an enema employed immediately after a meal may produce dyspepsia in a person unaccustomed to such a proceeding. Flatulent distension of the colon, with borborygmi, is also a frequent effect of dyspepsia.

Besides these symptoms, referable directly to the digestive organs, there is a general depression of all the vital powers. The patient complains of a sense of languor and weariness of the limbs, especially after his meals. He is melancholy, morose, or irascible. His circulation is feeble, his pulse soft and compressible, slow when he is at rest, but quickened by any exertion. Palpitation is commonly complained of: and sometimes the heart's action becomes intermittent. The sense of dyspnœa, already referred to, may lead him often to sigh, or to draw a deep breath. The skin is flabby and moist; there is no fever; the complexion is often pallid, sallow, and muddy. There may be marked anæmia, but not usually, and loss of flesh. The urine is copious and clear. Such, according to Dr Wilson Fox, is the common clinical history of atonic dyspepsia.

In *chronic gastric catarrh* the symptoms are in most respects very similar. Even in this affection the patient seldom complains after his meals of anything amounting to pain; and tenderness of the abdomen, although often present in some degree, is not usually marked. On the other hand, thirst is a very prominent and distinctive symptom, especially in the intervals between meals. The patient often experiences a sense of exhaustion or of internal heat, which is relieved by drinking. The appetite is capricious. The breath is often offensive. A nasty taste in the mouth is often complained of, especially on first rising in the morning. The gums are spongy, red, and inclined to bleed. An excessive secretion of saliva is not uncommon, and at night it may escape from the mouth, wetting the pillow. The tongue is often of a bright red colour, and raw-looking, the papillæ standing out as bright red points. Or this condition may exist only at its sides and tip, the rest of its surface being coated with a white or brownish fur of greater or less thickness. The lips are often dry and cracked. The mucous membrane of the pharynx may be granular and inflamed, and it may secrete a tenacious mucus which is a source of great annoyance and discomfort to the patient.

Emaciation is said to be almost constant in chronic catarrh of the stomach. Slight febrile disturbance, preceded by rigor and malaise, is very common. Sometimes it occurs at night, and then it is often followed by copious perspirations during sleep. The urine is generally scanty and turbid, depositing urates or oxalates; or sometimes it is alkaline, throwing down phosphates. The expression is anxious and careworn.

Vomiting does not necessarily occur in chronic catarrh of the stomach any more than in atonic dyspepsia. Even nausea is generally not much complained of. In the dyspepsia of habitual drunkards, however, vomiting of mucus, especially in the morning, is one of the principal symptoms, and it is probable that this kind of vomiting is always an evidence of a catarrhal state of the gastric mucous membrane. Some caution appears to be required in concluding that vomited matters contain mucus from their naked-eye appearances only; Frerichs has shown that starchy substances are sometimes converted in the stomach into a tenacious glutinous material which may resemble mucus very closely.

In some dyspeptic patients in whom vomiting occurs with considerable frequency the ejecta are intensely sour. This is generally due, not to an over-secretion of acid by the stomach, but to the formation of lactic, butyric, and acetic acids by fermentation from the starchy and saccharine elements of the food. These acids are often developed with great rapidity, and in such large quantity that when the patient vomits the throat burns, the teeth are set on edge, and the eyes smart, just as though strong acetic acid had been taken into the mouth. At the same time the sour smell of an acid, volatile at a low temperature, is diffused through the air. A further evidence that fermentation is the cause of the formation of acid in such cases is the fact that gas is evolved, which (according to Dr Wilson Fox) consists of a mixture of carbonic acid and volatile carbohydrogen. Sulphuretted hydrogen is also found whenever eggs or other articles of diet containing sulphur have been eaten in considerable quantity.

The *morbid appearances* presented by the mucous membrane are described as being different in atonic dyspepsia and in chronic gastric catarrh respectively. In the former the lining of the stomach is thin and transparent. In the latter it is almost always thickened and indurated. It may even be so tough that it can be stripped off the subjacent tissue in large pieces, or the submucous tissue may at the same time be white and fibrous, in which case there is increased rather than diminished difficulty in separating the coats. Near the pylorus the mucous membrane is often mammilated in cases of chronic gastric catarrh. But it is important to note that this condition is not necessarily the result of inflammation, for it may be found even in a healthy organ, being then caused by the contraction of the muscular layer which exists round the bases of the secreting glands. The most characteristic change in the appearance of the interior of the stomach in cases of chronic catarrh is, however, its ash-grey pigmentation. This, when closely examined, is seen to depend upon the presence of numerous minute specks scattered thickly over it. Under the microscope they are seen to consist of granules of pigment (doubtless originally derived from hæmatin) which are deposited in the connective tissue between the tubes or even in the epithelial cells.

The gastric glands appear to present morbid changes in both forms of chronic indigestion. At least it seems to be certain that such changes are often observed in cases which in all other respects would come under the head of atonic dyspepsia. The secreting tubes are then found to be shrunken and wasted, and to have undergone fatty degeneration. They are often irregular in form and calibre. Their epithelium may have almost entirely disappeared, being represented only by granular *débris* and by fat globules. In chronic gastric catarrh the changes in the epithelium appear to be the same, but according to Dr Wilson Fox the membrana limitans may be thickened in this condition. Cysts are not uncommonly found which probably arose by the distension of parts of the tubes that had become constricted off from the rest. Dr Wilson Fox observes that fatty degeneration is especially apt to affect groups of the glands one or two lines in diameter, which are then visible as small dead white spots in the mucous membrane. Dr Habershon, Dr Handfield Jones, and Dr Fenwick are the other observers who have worked most at this branch of morbid histology. Several of Dr Jones's cases were in persons advanced in life, so that the fatty changes in the tubes might be attributed to a " senile degeneration." Dr Wilson Fox asserts that the gastric glands may be replaced by a fibro-nucleated tissue in atonic dyspepsia, but this appears to me hardly probable, and, as he himself remarks, simple atony is often itself the result of a pre-existent inflammation. Dr Fenwick has demonstrated the fact that when the secreting tubes are atrophied the digestive power of the dead mucous membrane is much less than under normal conditions. He found changes in the glands especially frequent in those who died of cancer of the breast, and he is disposed to regard this as the cause of the rapidly increasing anæmia which occurs in such cases.

I have not yet mentioned redness of the stomach as one of the appearances characteristic of catarrhal inflammation, and, indeed, in many cases of this kind there is no redness, at least in the dead body. But in some forms the most intense injection of the gastric mucous membrane is an almost constant appearance. This is particularly observable when there is chronic obstructive disease of the heart. The stomach is then found lined with a thick layer of mucus, and after this is washed away its surface is seen to be of the most vivid crimson colour, which may either affect a large part of its surface uniformly or occur in spots. Ecchymoses are often present at the same time, and still more frequently they are simulated by small patches, due to arborescent injection of the branches of some minute vessel. When effusion of blood occurs into the submucous tissue it would seem that the gastric juice sometimes dissolves off the corresponding part of the mucous membrane. A little ulcer is the consequence, the floor of which is occupied by a layer of black coagulum. This process is known by the name of "*hæmorrhagic erosion.*" Another form of gastric catarrh in which the stomach is often found intensely reddened is that which results from alcoholic intemperance. An unskilled pathologist may easily be led to suspect the presence of an irritant poison in cases of this kind. Some time ago I made an autopsy in a case of a young man who had suddenly died in a railway train early in the morning. I could discover no cause for his death, but the stomach was most intensely reddened and ecchymosed. It was clearly ascertained that there had been no foul play, and there appeared to be little doubt that the abuse of stimulants had been the cause of the gastric irritation.

The *causes* of atonic dyspepsia and of chronic gastric catarrh are very similar. According to Dr Wilson Fox the disposition to the former is frequently inherited, and he says that this is also to some extent the case with the latter. Age is an important element in the causation of atonic dyspepsia; as life advances the digestive power of the stomach undergoes diminution. Hot seasons, relaxing climates, exhausting discharges, sedentary occupations, venereal excesses, prolonged anxiety of mind, long continued depressing emotions, are also enumerated as being capable of setting up the affection in question.

Other more obvious causes of indigestion are the habit of waiting too long between the meals (Dr Fox has seen several cases in which it was due to fasting from an early breakfast to a late dinner), imperfect mastication of the food (the state of the teeth should always be looked after), taking too much fluid (especially warm fluid) with the meals, the abuse of condiments or of tea or coffee, or excessive smoking, the taking bodily exertion or making mental efforts while digestion is going on.

The quality of the food may also be concerned. Dr Chambers cites the case of a poor needlewoman who had subsisted for a year on bread, potatoes, and tea, getting sometimes a little bacon but hardly ever other kinds of meat, and who suffered so much from dyspepsia that she dreaded to eat. In other cases indigestion always follows some particular article of diet, such as fatty matters or soups. Dr Chambers has attempted to describe "indigestion of vegetable food," "of albuminoid food," "of fatty food," and "of watery food" separately, but I think with no great success. One curious instance that he gives is that of a lady who from childhood had never been able to take roast beef without afterwards having heartburn. This Dr Chambers attributes to the fat which lies between the muscular bundles in that kind of meat. With regard to digestion, however, many idiosyncrasies are met with, of which it is not possible to give explanations, but which the physician must not ignore.

The *diagnosis* between atonic dyspepsia and chronic gastric catarrh is often unsatisfactory; it must be based upon a careful analysis of the symptoms. But, after all, it is of little consequence, except for the circumstance that these symptoms individually require different kinds of treatment, and with a little practical experience one probably learns to adjust one's remedial measures to the necessities of the case as well without attempting to draw what is often a very fine distinction between affections that are really closely allied to one another.

The diagnosis between chronic dyspepsia and the serious organic diseases of the stomach is of infinitely greater importance. I will leave it to be discussed after they have been described; but I may say in this place that their early symptoms are very commonly attributed to mere indigestion, and that the possibility that one or other of them may develop itself must never be overlooked in any case that seems to be protracted or severe. Vomited matters should always be subjected to the closest scrutiny, and we must make it a rule to examine the patient in the recumbent posture, and with the surface of the abdomen exposed to view and manipulation. I can never forget the case of a gentleman in whom, as soon as his shirt was raised, the existence of obstruction at the pylorus was indicated by the peristaltic movements of a dilated stomach which were at once visible, but who assured me that his abdomen had not hitherto been examined, although he had been under the care of more than one specialist.

The *prognosis* in dyspepsia depends chiefly on whether its causes are or are not capable of being completely removed, on the age of the patient, and on the degree of severity and duration of the symptoms, from which one must consider whether or not the structure of the gastric glands is likely to have already become impaired. Proper treatment scarcely ever fails to give some relief; most cases are very greatly benefited, but it seldom happens that a case of long standing is permanently and absolutely cured.

In regard to the *treatment* of these affections, it will be most convenient to take first chronic catarrh of the stomach. For this affection sedatives are the remedies that should be first employed, and especially bismuth. A drachm of Schacht's solution, or ten grains of the subnitrate, may be administered three times daily. At the same time, small doses of some alkali, and of morphia may be given with great advantage; a very useful formula is the Mist. bismuthi sedativa of the Guy's pharmacopœia.* Another valuable remedy in cases of this kind is magnesia; it may be prescribed with three-minim doses of dilute hydrocyanic acid, and equal parts of lime-water and cinnamon-water. Again, the oxide of zinc (in doses of two to three grains) is of service, especially when the cause of the gastric disorder is alcoholic intemperance. In long-standing cases, Dr Wilson Fox also recommends the nitrate of silver (in doses of a quarter of a grain to a grain), the oxide of silver (in doses of one grain to two grains), alum (in doses of two to five grains), tannin or decoction of oak bark, and matico. This writer gives a caution against the use of purgatives in cases of chronic irritative dyspepsia; which, he says, "are often aggravated by a persistence in their use." He advises that, when necessary, the action of the bowels should be solicited by the daily use of enemata of cold water, although he allows castor oil in some cases, and in others the decoction of aloes, or a pill containing aloes and extract of nux vomica, which is to be taken with the food.

Among the natural mineral waters of Great Britain, the most useful in cases of chronic gastric catarrh, are said by Dr Fox to be those of Harrogate, Bath, and Leamington. Trousseau recommends Plombières and Bagnères de Bigorre, in France; as well as Vichy and Pougues.

On the other hand, in atonic dyspepsia, the treatment must be such as will tend to restore the functional activity of the stomach. The most important point of all is the regulation of the diet. The number of the meals, and the quantity of food taken at each meal, should be carefully adjusted, according to the idiosyncrasy of the patient. As a rule, there should be three meals daily; at two of which freshly cooked meat should be eaten. Beef and mutton, poultry and game (but not hare or rabbit) are to be recommended; pork and veal, and salted or preserved meats are to be forbidden. Eggs agree well with some dyspeptic patients, whereas others are unable to take them. Dr Wilson Fox says that fish is less wholesome than meat in cases of this kind. Vegetables must not be omitted from the dietary, but are to be eaten with caution. Potatoes should always be taken sparingly, if at all; they must be well boiled and not young. Among other vegetables Dr Wilson Fox says that turnips, parsnips, Jerusalem artichokes, onions, and the cruciferæ often disagree; but that spinach, vegetable marrow, beetroot, young peas, and French beans may commonly be taken in moderation. According to Dr Chambers, however, stewed lettuces, cabbages, watercress, and salad may be taken; while he says that peas and beans are famous for causing flatulence. When vegetables are found to disagree, their place may be supplied by rice or maccaroni, and such fruits as stewed prunes, grapes, and strawberries. New bread should never be eaten by persons who are subject to indigestion; "aerated bread" is in many cases to be preferred to that made with yeast. Sometimes it is of great importance to substitute biscuits for bread. Light farinaceous puddings generally agree well with dyspeptic patients. Pastry is to be strictly avoided; but according to Dr Chambers, "short" pastry (in which the butter is thoroughly incorporated with the dough) can sometimes be eaten with impunity. Sugar, says Dr Fox, "may be used in moderation;" but Dr Chambers objects to its being taken "in such quantity as to cause a sweet taste." Everyone is agreed that lobsters, crabs, nuts, pickles, and cheese are to be strictly forbidden. A large amount of fluid should not be drunk at meals. Cocoa, or milk and water, may be used as substitutes for tea or coffee. Dr Fox advises that a moderate quantity of wine (sherry, claret, hock or champagne) should be taken twice daily; but I think that the more usual practice is to limit the patient to weak whiskey or brandy and water with his meals. Malt liquors are certainly injurious. Food is to be taken slowly, time being allowed for mastication and the due admixture of saliva; and on this account it is advisable that the patient should have his meals in company with other persons.

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* It must be kept in mind that the preparations of bismuth, like those of iron, give a black colour to the fæces. Dr Bristowe thought that they also sometimes produce a line on the gums resembling that caused by lead. But I think that this certainly was a mistake. His account of the appearance which he had observed in one or two instances is that the line was "bluish-red," "wider and redder" than the lead line, a description of which will be found below at p. 162.

General hygienic treatment is of great importance in cases of atonic dyspepsia. The patient should retire early to rest, and sleep in an airy room. During the day he should be in the open air whenever he can, and he should take as much exercise as possible, short of fatigue. Riding on horseback is often to be recommended, and for some patients yachting, or a sea voyage. A daily tepid bath is generally advisable, with the use of the hair glove or flesh brush. Cold baths are to be taken only when they are followed by good reaction. Dr Wilson Fox speaks of Brighton as the best place for persons affected with this form of dyspepsia; next in order he places Scarborough, Dover, Folkestone, Margate, Eastbourne, Malvern, Tunbridge Wells. He also speaks highly of Ilfracombe. The patient's residence, he says, should be in "a high situation on a porous soil."

The medicinal treatment of atonic dyspepsia consists mainly in the administration of remedies which tend to assist the process of digestion. Among these an important place belongs to the alkalies, which have been shown by Blondlot and Bernard to increase the secretion of gastric juice. One of the simple or aromatic bitters may often be given at the same time; such as hop, chamomile, or calumba. A very useful formula is one which contains carbonate of soda, rhubarb, and calumba. Dr Wilson Fox speaks of gentian as requiring to be used with some caution, lest it should irritate the stomach. Nux vomica is exceedingly useful, but Dr Fox says that it should not be taken continuously for more than a month or six weeks, on account of its liability to produce irritation of the spinal centres. Dr Chambers alludes to a case in which it produced a painful irritability of both mind and body after each dose. I remember one patient who said that this drug made his legs start so that he could not walk over London Bridge; but I think that he was taking grain doses of the extract; in doses of a quarter of a grain I have never seen it do any harm. I commonly prescribe it in the form of a pill, with a grain of sulphate of iron and a grain of extract of aloes or of compound rhubarb pill, to be taken just before one or more of the meals.

In many cases of atonic dyspepsia, and particularly in the later stages of the affection, the dilute mineral acids are very useful, especially the hydrochloric acid. In doses of fifteen or twenty minims, properly diluted and taken with or after the meals, it prevents the sense of weight and oppression which would otherwise be experienced by the patient, and it is said to prevent acidity and flatulence arising from fermentation of the food consequent upon imperfect action of the gastric juice. Trousseau speaks very highly of this remedy, which appears to be but little known in France. He mentions that he learnt its use by sitting at dinner next to a tourist who said that he never travelled without a little bottle of the acid, of which he took a few drops after each meal. Pepsine, again, is often useful in cases of atonic dyspepsia. But Dr Pavy has shown that care is required in obtaining it at the druggists, since much of what is sold in London is devoid of any active properties. A plan which this physician has proposed, consists in digesting meat artificially (by means of an infusion of dried pig's stomach, acidulated with hydrochloric acid) before it is swallowed.

General tonics are also very serviceable in these cases, but even the milder preparations of iron (although indicated when the patient is anæmic) are said sometimes to disagree with dyspeptic patients, and according to Dr Fox, the same is the case with quinine, particularly if it be given with sulphuric acid.

I have still to mention certain effects of gastric disorder, which although they are sometimes associated with the ordinary symptoms of dyspepsia, yet often occur by themselves, and may even reach such a degree of intensity that they have to be regarded as independent affections.

GASTRALGIA.—Perhaps the most important of these is *pain*. I have already mentioned this as one of the symptoms of dyspepsia, particularly in its inflammatory form; but it also very frequently occurs unattended with any indication of impairment in the digestive process. Several names are applied to pains situated in the stomach, but unfortunately different writers use them in different senses. Cullen employed *cardialgia* for the less severe varieties which would commonly be called "heartburn" or "acidity," while he described as *gastrodynia* a more violent but also more transient pain, such as would usually be spoken of as "cramp" or "spasm" of the stomach. Most English writers follow Cullen in the use of these names, but the Germans employ them with meanings exactly reversed. *Gastralgia* is a term which is used chiefly by the French, and with a very wide range of application. The late Dr Anstie proposed to limit it to a particular kind of pain, namely, to that which comes on when the stomach is empty, half an hour or so before the time appointed for a meal.

This last kind of gastric pain is mentioned by all writers on the subject. It is often quickly relieved by even a small quantity of food. Sir Thomas Watson mentions the case of a clergyman whom he knew, and who was much harassed by its recurrence several times daily until he found by accident, after having tried a round of drugs, that eating a small biscuit would at once appease it. This writer also says that a drachm of the aromatic spirits of ammonia, or half a drachm of magnesia, will sometimes remove the pain in a moment as if by magic. According to Dr Budd, the gastric pain which occurs when the stomach is empty is also accompanied by slowness of the pulse and by coldness of the surface of the body; the recumbent posture helps it away; hydrocyanic acid is the medicine which he recommends for it. On the other hand, Dr Anstie regarded it as a form of neuralgia, and he speaks of strychnia as the most valuable remedy for it. He prescribed five or ten minims of the tincture of nux vomica three times a day, or sometimes gave $\frac{1}{80}$ to $\frac{1}{50}$ of strychnia by subcutaneous injection. One case in which this remedy effected a cure was that of a patient who had actually attempted suicide on account of the agonizing pain which he had to endure. Arsenic was also recommended by Dr Leared for this affection. I have occasionally made trial of it, but without any marked success.

When gastric pain comes on *after* food it may be due to a variety of causes. Its diagnosis always requires great care, for the pain arising from organic disease of the stomach is usually of this kind. The strongest indication of the presence of such disease is the circumstance that the pain begins as soon as food is taken, and lasts until digestion is completed or until vomiting occurs. But, as we shall presently see, these characters may be wanting. On the other hand, very severe and protracted pain is sometimes complained of after every meal by nervous or hysterical persons, in whom there is no serious disease.

Another form of gastric pain—I believe first described by Dr Abercrombie—begins from two to four hours after a meal, and lasts for several hours. This writer thought that its seat was in the duodenum. But Sir Thomas Watson points out that one can generally remove it by giving an alkali, or by letting the patient swallow a cup of warm tea. He therefore supposes that it is due to the continued secretion of gastric juice after the food has passed through the pylorus, and then adds that the onset of the pain may often be prevented by a small quantity of alkali in some aromatic water taken immediately after dinner. Trousseau speaks of this kind of pain as being often attended with a sense of sinking at the stomach, and a craving appetite, and a great feeling of weakness. Constipation usually accompanies it, but sometimes diarrhœa, which he attributes to the circumstance that in cases of this kind the food is propelled into the duodenum before the digestive action of the stomach is completed. The treatment which Dr Abercrombie found most useful consisted in giving two grains of sulphate of iron, with one grain of aloes and five grains of aromatic powder, three times daily. The sedative medicines enumerated at p. 123, are also useful in such cases.

But pain in the neighbourhood of the stomach, arising from disorder of that viscus, sometimes bears no relation whatever to the times at which the meals may be taken, or to the stage which the process of digestion may have reached. I have several times seen pain in the left side, under the nipple and running round to the shoulder, relieved instantly by bismuth and morphia, although I had been unable to elicit a single fact indicative of its having been due to gastric irritation. For this kind of pain—coming on at uncertain intervals in most violent paroxysms—Cullen reserved the name of "gastrodynia." Sir Thomas Watson says that it is often accompanied by a sensation of distension, much anxiety, and extreme restlessness. In females hysterical symptoms are frequently present. Sometimes the stomach is distended with enormous quantities of gas.

Gastrodynia may recur at irregular intervals for a very long period without appreciably affecting the general state of the patient's health. It cannot in such cases be due to any active disease. Probably it is sometimes the indirect result of pathological changes long since come to an end. Bamberger speaks of the cicatrices of gastric ulcers as giving rise to paroxysmal attacks of pain, and it seems exceedingly probable that they may do so by irritating the filaments of nerves embedded in them. Not long ago I made an autopsy in the case of a lady who had for years suffered from a pain in the back, which had been supposed to be connected with an abscess near the sacrum she had had when a child. I found all the parts in front of the spine—the aorta, the vena cava, and the nerves—embedded in a dense mass of cicatricial fibrous tissue. This had doubtless been in some way the cause of the pain. I shall hereafter have to mention another case in which the destruction of an hydatid in the liver was followed by severe pain, evidently due to interference with nervous filaments during the contraction of the cyst.

On the other hand, a person may suffer from gastrodynia for many years and yet if one should have an opportunity of making an autopsy one may find nothing to account for it. Bamberger gives a case of this kind. It occurred in a powerful man, who for nine years had been subject to frequent attacks of the most violent pain in the stomach, lasting for days, or even weeks, and attended with great prostration and temporary loss of flesh. He died of acute phthisis. A slight dilatation of the stomach was the only morbid change in that organ.

Gastrodynia has to be distinguished from several other kinds of pain. In the first place pain arising in the colon may resemble it somewhat closely; this I leave to be discussed when I am speaking of colic (*infra*, p. 159).

Again, according to Briquet, the abdominal muscles are often the seat of pain, without any affection of the subjacent parts. He lays stress on the circumstances that superficial tenderness is present, that the left recti and obliqui abdominis are those principally affected, that not only their fleshy parts but also their tendinous attachments are concerned, and that dorsal pain and tenderness in the vertebral groove often exist at the same time. But we shall hereafter see that rigidity of the upper part of the rectus with tenderness is a very common effect of organic disease of the stomach, and I do not know why it should not occur even when the pain is of functional origin. I remember, however, one very striking instance in which a pain in the left hypochondrium, which had long resisted other treatment, was again and again removed by quinine and iron, and in that case the pain was probably myalgic. Rheumatic pain in the abdominal muscles would be recognised by its being increased by movement of the body.

Lastly, pain situated in the epigastrium, over the stomach, may be continuous, and may last for a great length of time; but of this kind of pain I think one may say that it is comparatively seldom caused even by gastric disease, and perhaps never by mere gastric disorder. There are doubtless many conditions that may give rise to it; but two in particular must always be borne in remembrance,—aneurysm of the abdominal aorta and incipient disease of the dorsal vertebræ. Some very striking instances in which epigastric pain was due to spinal disease were related by Mr Hilton. One was that of a boy who for two months had been complaining of severe pain just above the pit of the stomach, and who used to walk about with his hands placed over that region, and with the body a little inclined forwards. It seemed as though he were suffering from irritation of some of the abdominal organs, and he had been treated on that supposition, but without much benefit. The pain was relieved when the boy lay down. Its seat was not to one side of the body more than to the other. Disease was detected between the sixth and seventh dorsal vertebræ and pressure on their spines excited the pain in front. He was kept in a recumbent posture for four or five months, at the end of which time he was completely cured. Another case was seen by Mr Hilton with Dr Addison. A Westminster boy had pain at the pit of the stomach and occasional vomiting. He was found to have disease between the same two vertebræ; he too was easy when lying in bed. He was made to lie down almost uninterruptedly for two or three months, and from that time he got well.

True gastrodynia is often very intractable. Sir Thomas Watson recommends the application of a mustard poultice to the epigastrium, and the administration of a carminative (such as a few drops of cajeput oil suspended by mucilage) or of sedatives, among which he assigns the chief place to hydrocyanic acid.

Anorexia.—Alterations of the appetite are commonly due to gastric disorder. Loss of appetite (or *anorexia*, as it is sometimes called) may indeed be a symptom of almost any kind of disease. But it doubtless depends upon the loss of digestive power which accompanies so many morbid conditions, particularly those attended with fever. Dr Beaumont found that when Alexis St Martin was feverish, the secretion of the stomach was diminished or suppressed, and food remained undigested for twenty-four or even forty-eight hours. Hence it is that patients suffering from acute diseases do not have meals, and are allowed only fluid nourishment, in small quantities and at intervals which are often extremely short. In other persons the appetite may be greatly improved by the administration of tonics; but such medicines should never be prescribed until it has been ascertained that the anorexia is not due to disorder of the digestive organs, which must be first corrected. Excessive appetite, on the other hand, is called *bulimia*. I shall subsequently have to refer to it as being produced by the presence of worms in the intestines, as a symptom in diabetes and in some diseases of the mesenteric glands. I have already mentioned it as accompanying one of the forms of pain in the stomach.

Lastly, *pica* is the name given to a perverted state of the appetite, in which substances that have no nutrient properties are swallowed greedily, or filthy matters, such as human excrement, horsedung, &c.

Regurgitation.—Other symptoms of gastric disorder are those which consist in the regurgitation of gaseous or fluid matters upwards through the œsophagus. Of these the simplest form is *eructation* or belching. Generally gas alone arises, but sometimes a small portion of imperfectly digested food, which, however, is instantly carried back into the stomach. In rare cases a large part of the food is habitually brought back into the mouth, to be deliberately remasticated and swallowed a second time. Some years ago I saw a patient of Dr Pavy's who possessed this habit of ruminating. Dr Copland collected a number of cases of this kind, some of which came under his own observation. In one patient the rumination began in from fifteen minutes to an hour after almost every meal. Each bolus of food came up during an act of expiration; it had the same taste and flavour as when first swallowed; there was neither nausea nor pain; he masticated it a second time with pleasure. I do not find that any treatment is applicable to persons who ruminate, beyond advising them to eat very slowly and with moderation.

A somewhat analogous affection is what is called waterbrash or *pyrosis.** This is not very common in England, but the people of Scotland are very liable to it, and still more so those of Norway, Sweden, and Lapland. It is supposed to be caused in the Scotch by the oatmeal of which they eat so largely. Dr Cullen, who was very familiar with pyrosis, described it as coming on usually in the morning when the stomach is empty. According to him, it begins with a severe pain at the pit of the stomach, which, after continuing for some time, brings on the eructation of a thin watery fluid in considerable quantity. The fluid is sometimes acid, but is often absolutely tasteless. The repetition of the eructation seems at length to give relief to the pain, and the attack is then at an end. It is apt to return more or less frequently for a considerable length of time. Cullen says that the complaint occurs chiefly among the lower classes, in women more often than in men, and between puberty and middle age rather than at any other period of life. He speaks of it as often unattended with any symptoms of dyspepsia. In England, however, pyrosis comparatively seldom appears as an independent malady, apart from other effects of gastric disorder. Sir Thomas Watson speaks of one remarkable case in which no less than three pints of a thin tasteless liquid were brought up every day. Writers differ with regard to the nature of this secretion. Some, with Dr Handfield Jones, regard pyrosis as a catarrhal affection of the gastric mucous membrane, analogous (for instance) to bronchorrhœa. But the liquid is often ejected without any effort of vomiting; and, moreover, this sometimes occurs immediately after, or even during, a meal. Dr Chambers gives the case of a retired surgeon, who often had to leave the room at mealtimes, and would throw off as much as five or six ounces of frothy clear liquid having a cold taste. The contents of the gastric cavity never came up at the same time, although the ejection of the fluid sometimes made him retch. There seems, therefore, to be much probability in the opinion suggested by Dr Chambers that the fluid is really saliva, which trickles down the œsophagus, and, being arrested by spasm of the cardiac orifice, collects there until it gushes back into the mouth. Frerichs, indeed, is said to have detected sulpho-cyanide of potassium in liquid of this nature. One may perhaps object that Dr Pavy found it to have the power of digesting, but evidently this might have been due to the admixture of a little gastric juice. The last-named observer showed that water injected into the stomach of an animal quickly becomes charged with pepsine, so that if the possession of digestive power should create any difficulty in the application of Dr Chambers's theory, this might be removed by supposing that the saliva had passed down into the stomach before being ejected. Dr Pavy, however, himself thinks that the fluid is often secreted by the stomach.

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* Etymologically, pyrosis (πῦρ = fire) should mean the same as heartburn; but in England it is never used in this sense.

I have often seen the preparations of bismuth very useful in the treatment of pyrosis. Sir Thomas Watson recommends opium combined with an astringent, as, for instance, in the pulvis kino compositus. Dr Pavy is in the habit of prescribing the liquor opii sedativus in eight-minim doses, with an ounce of the compound infusion of gentian, three times a day.

Vomiting.—Another effect of gastric disorder is *emesis* or vomiting. This has already been mentioned as occurring in certain forms of dyspepsia, and we shall subsequently find that it is a principal symptom of all the more serious organic diseases of the stomach. But it may also be met with independently of all these conditions.

The act of vomiting is usually preceded by a peculiar feeling which is termed *nausea* (literally " sea sickness "). And, in persons unaccustomed to being sick, this is perhaps always accompanied with sensations of giddiness or faintness, coldness of the surface, and pallor of the lips and face, and with a small and feeble pulse. After some seconds, or a few minutes, retching occurs; and this is followed by the expulsion of the contents of the stomach. But it is by no means always the case that vomiting is preceded by such painful sensations and efforts. Some persons, chiefly women, suffer from year to year from occasional sickness, which in them may be attended with scarcely any discomfort. This kind of vomiting is especially apt to occur at night or in the early morning. And so far it resembles the chronic vomiting of those who indulge in alcoholic stimulants to excess. But it is a mistake to suppose that morning sickness is necessarily an indication of such intemperance.

Sometimes habitual vomiting, independently of gastric pain and of any indication of dyspepsia, may reach such a point as to be alarming and even dangerous to life. In hospital practice I have seen several cases of this kind. Some striking instances are related by Dr Chambers. In one of them the affection had been of three years' duration, and it was stated that the food was always returned, unchanged in appearance, within ten minutes after being swallowed. Another patient was said for five years to have hardly ever kept down a whole meal. This kind of sickness is almost confined to the female sex, and occurs chiefly in young women. It is very frequently associated with disorder of the menstrual functions; in one of Dr Chambers's cases, to which I have just referred, it was attributed to a chill by which the catamenia had been suppressed for several months. These patients too are often hysterical. They sometimes reject the food before there has been time for it to be swallowed. According to Dr Chambers the tendency to vomit can sometimes be checked by a strong effect of the will. He also notices that a patient suffering from this kind of sickness sometimes communicates the complaint to a neighbour of the same sex and age. But the most remarkable feature of many of these cases is that although the vomiting is so constant, yet there is little or no loss of flesh. It is evident that a good deal of food must in reality be retained by the stomach.

In cases of this kind the *diagnosis* cannot be carried further than "irritability of the stomach," or "hysterical vomiting." But it is of the most extreme importance that the practitioner should remember that there are several organic diseases—affecting parts far distant from the stomach—of which a similar form of vomiting may be one of the symptoms. There is indeed hardly any morbid condition in which the stomach does not occasionally reject its contents. But the point to which I now wish to draw attention is that in certain diseases irritability of the stomach is often the earliest and for a time the only indication that the patient is ill. The most important of these is perhaps incipient pulmonary *phthisis.* The lungs should always be most carefully examined before one arrives at the conclusion that habitual vomiting is mainly due to functional disturbance of the stomach. And, if there be any other ground for thinking that tubercular disease is likely to develop itself, a guarded opinion should be given even though there may be no discoverable stethoscopical evidence of its presence. Another morbid state, of which vomiting is a principal symptom, is that due to *Addison's disease* of the suprarenal capsules. Again the possibility of the supervention of *cerebral disease* must never be overlooked; I have seen cases of abscess of the brain in which there were few other symptoms besides sickness. According to Romberg, the vomiting which accompanies affections of the brain is characterised by the absence of nausea and of retching, and by its occurring when the head is moved, as in swinging, shaking, or stooping, or in suddenly rising; it also occurs when the patient is erect rather than when he is recumbent. Writers generally state that affections of the spine seldom cause gastric disturbance, but I remember one case of obstinate vomiting, which was attributed to irritation of the roots of the splanchnic nerves by disease of the upper dorsal vertebræ; but I think that the patient afterwards proved to have likewise early phthisis. In female patients the possibility of *pregnancy* must never be forgotten, particularly if the vomiting should be only of a few weeks' duration. All these varieties of sickness are commonly attended with constipation. When chronic vomiting and diarrhœa occur in the same case, the presence of some form of *Bright's disease* of the kidneys should always be suspected. I have seen at least two instances in which the observance of this rule has led to a correct diagnosis; and in one of them this might not otherwise have been made, for the quantity of albumen in the urine was very small, and would probably have been overlooked. Another possible cause for the existence of vomiting and diarrhœa in the same patient is chronic *poisoning* by small doses of arsenic or antimony.

The diagnosis of "irritability of stomach" having been arrived at, its *treatment* is often highly successful. In many cases the best plan is to give the stomach entire rest for two or three weeks, the patient being fed solely by enemata. Or minute quantities of milk may be administered by mouth, as in the well-known case related by Dr William Hunter. A boy was brought to him in a state of the most extreme emaciation, who vomited up almost everything that he swallowed, in spite of the treatment of three very eminent physicians. Dr Hunter recommended that only a single spoonful of milk should be given at a time. The boy was never sick afterwards; he gradually became able to take more and more nourishment, and he ultimately recovered entirely. A most graphic and interesting account of the case is given in the sixth volume of Dr Hunter's 'Medical Observations and Inquiries.'

The application of a blister to the epigastrium is often highly serviceable in cases of this kind, particularly if it be kept open by an Albespeynes' plaister, or in some other way. The sedatives recommended at p. 125 may often be prescribed with advantage, but particularly morphia or opium. I remember one case in which the oxalate of cerium (in doses of one to two grains) succeeded, although other remedies had failed. Creasote is sometimes serviceable. In the cases related by Dr Chambers a daily shower-bath appeared to do much towards the restoration of vigour of mind and body, and in some of them the preparations of valerian were useful.

Lastly, gastric disorder may manifest itself by producing reflected disturbance of other parts. Thus *palpitation* of the heart and *cough* are occasionally produced by overloading of the stomach.

A somewhat similar affection is hiccough or *singultus*. This is due to a sudden spasmodic contraction of the diaphragm, repeated at more or less regular intervals and attended with a clicking sound which is caused by the abrupt passage of air through the glottis. Its recurrence can often be stopped by holding the breath. Hiccough is not usually a matter of any consequence, and lasts only a few minutes, or at most an hour or two. But in dangerous illnesses it sometimes continues for days together, and it may then exhaust the patient and appear to be the immediate cause of death. In all probability it is due to the presence in the stomach of food with which that organ is incapable of dealing.

No one can make frequent autopsies without observing how often the stomach contains a pint or more of brandy and egg mixture or of some similar substance, which must include all that had been given by the nurses for some hours before death. I remember at least one case of fever in which, as the end was approaching, the relatives began to flatter themselves with vain hopes that the patient would recover because he took all the nourishment that was offered to him, but this evidently was not absorbed, for I could constantly produce a splashing sound by manipulating the upper part of the abdomen.

Dr Edward Liveing mentions that he had a man past middle age under his care in whom hiccough occurred in paroxysms of twelve hours' duration about twice a week for four years, and he quotes a case of Dr Prichard's, of a little girl of twelve, who for nearly three years was subject to fits of violent hiccough, even during sleep, which lasted from ten minutes to an hour and returned three or four times during a day and night. She was cured by turpentine.

HÆMATEMESIS.—I have already spoken of matters of various kinds as being sometimes ejected from the stomach, but I have not specially mentioned vomiting of blood, or, as it is called, hæmatemesis. For this is, in the majority of cases, the result of some serious lesion, although it may at times occur independently of any such cause. This, therefore, appears to be the most fitting place to discuss it before I pass on to describe the organic diseases of the stomach.

Diagnosis.—In investigating a case in which blood is said to have been vomited, one must, in the first place, make sure that the blood was really rejected from the stomach. Strange as it may appear, there is often considerable difficulty in distinguishing between hæmoptysis and hæmatemesis, and particularly so when one has to rely upon the statements of the patient and does not see the blood actually brought up. When blood escapes into the air-passages in large quantities it may issue from the mouth in gushes; and, as Sir Thomas Watson points out, some of it may pass back into the pharynx and there excite retching and vomiting. Indeed, a portion of the blood may run down the œsophagus. I lately made an autopsy in a young child who died of hæmoptysis, and in whom an ounce of coagulated blood was found in the stomach. On the other hand, when sudden and profuse hæmatemesis occurs, the blood may irritate the larynx in passing over it, and so provoke a paroxysm of cough. The patient's statements, therefore, may afford a very unsafe basis for distinguishing between vomiting of blood and hæmoptysis; the diagnosis must be founded upon a consideration of the conditions under which these two forms of hæmorrhage severally occur. The stomach is a large cavity into which a great quantity of blood may ooze before it excites vomiting. Thus hæmatemesis is commonly *preceded* by the characteristic symptoms of hæmorrhage, pallor of face, dimness of vision, giddiness, or even fainting. On the other hand, hæmoptysis occurs quite suddenly, the patient finding the hot blood in his mouth or feeling a tickling in his throat, or a sensation as of bubbling in the chest immediately before he begins to cough up the blood. Again, after hæmatemesis, any blood that may be left in the stomach passes downwards, and (if not completely digested) it is ultimately discharged from the bowels. But when any part of the air-passages is the seat of the hæmorrhage, the blood that is almost necessarily sucked into the smaller bronchial tubes is afterwards got rid of by expectoration and the sputa remain discoloured by it for several hours or even for some days.

Again, the appearance of the blood after its ejection is generally different in cases of hæmoptysis and of hæmatemesis respectively. In the former it is usually frothy from admixture of air, it has an alkaline reaction, and it is of a bright red or "arterial" hue. In the latter it is acid, being mixed with the gastric juice. Dr Chambers mentions a case in which the application of test-paper to a woman's clothes over which she had thrown up blood, showed that it had come from her stomach. When the hæmorrhage is very profuse—and particularly if a large artery be opened—blood rejected from the stomach may be fluid and of a scarlet colour. But whenever it is retained for any length of time in the cavity before being vomited, it undergoes a peculiar change. It often coagulates, and sometimes solid masses of it are rejected which are so tough as almost to choke the patient. More frequently the blood, whether clotted or not, is acted on by the gastric juice, the acid of which turns it of a dark brown or black colour. The presence of comparatively small quantities of blood in vomited matters thus gives them an appearance like that of coffee grounds; while pure blood altered in this way resembles a black pudding or has more or less the appearance of tar.

Blood which passes down from the stomach into the intestines is altered in exactly the same way. It is often perfectly black (or as it is commonly called) "tarry." Evacuations having this character were supposed to consist of black bile by the older writers, who described them as characteristic of a special disease which they called *melæna.* But it has long been known that this affection is one of the results of hæmorrhage into the stomach, being, in fact, exactly equivalent to hæmatemesis. Dr Addison long ago taught that whenever blood having this black colour was discharged from the bowels the source of the bleeding was always the stomach. An exception to this rule ought perhaps to be made for the duodenum, although it is doubtful whether the blood discharged from an ulcer in that part of the bowel would be sufficiently long in contact with the gastric juice to undergo the peculiar change. But I quite believe that when the hæmorrhage occurs from the lower part of the intestine the blood is always more or less distinctly red. The dark appearance which the fæcal evacuations so constantly present when a patient has been taking a preparation of iron or bismuth medicinally must be distinguished from that due to blood. It is more of a slaty hue, and the fæces are usually remarkably dry. In any doubtful case one might apply the guaiacum test, which would doubtless at once reveal the presence of blood if the black colour were due to it.

It is important to note that hæmorrhage into the stomach may, and often does, occur without any blood being vomited. Thus a case was observed at Guy's Hospital in which a patient (who had once before brought up a large quantity of blood) became blanched, called out that she was dying, and did actually die in twenty-five minutes after a convulsive seizure. The stomach was full of clotted blood. I believe that other instances of a similar kind have presented themselves at the hospital, and one such is mentioned by Sir Thomas Watson. Doubtless therefore it is a frequent occurrence for small quantities of blood to escape into the gastric cavity without exciting vomiting. One can see no reason why small or even moderate quantities should ever be rejected unless there be also some other condition making the stomach irritable. And, up to a certain point, it is probable that they undergo digestion and are absorbed before they have passed down the whole length of the intestine. Thus the stools may themselves fail to afford evidence of the hæmorrhage. Still it is very important that whenever a patient's symptoms can possibly be attributed to bleeding into the cavity of the stomach, the fæcal evacuations should be most carefully examined.

A case in point is given by Dr Chambers. A woman, aged thirty-three, who had suffered from well-marked symptoms of gastric disease, grew rapidly weaker and paler, and her tongue became dry and furred, "as in hæmorrhagic fever," to use his expression. It was long suspected that she was passing blood, but she constantly denied that this was the case. She was made an in-patient, and then it was discovered that blood came from the bowels every time they acted.

On the other hand, hæmatemesis may occur without there being in reality any hæmorrhage from the stomach. Thus, in investigating a case in which blood is vomited, one must in the first place determine whether the blood comes from the surface of the stomach or from some other source. Attacks of hæmatemesis have sometimes been (so to speak) manufactured, the patients having first secretly drunk the blood which they afterwards vomited in the presence of others. Sir Thomas Watson mentions two cases of this kind. Again, when blood escapes into the back of the mouth or into the pharynx it is often swallowed by the patient unconsciously, particularly during sleep. And the first indication of the loss may be the occurrence of profuse hæmatemesis.

But in most cases of hæmatemesis the blood really comes from blood-vessels which run in or beneath the walls of the stomach. And we have now to consider what are the causes that may give rise to this form of hæmorrhage.

Causes.—Now, it is in the first place to be said that gastric hæmorrhage may occur in certain *general diseases*, as a consequence (it is supposed) of changes in the blood itself: for example scorbutus, purpura hæmorrhagica, malignant smallpox, yellow fever, acute yellow atrophy of the liver. Then, again, it it may be a subordinate symptom of obstructive disease of the heart. It is said, too, that hæmatemesis may be caused by disease of the spleen. Sir Thomas Watson quotes from Latour the case of a man whose spleen was immensely enlarged, as the result of obstinate ague. Latour foretold that hæmorrhage from the stomach would occur. His prognosis was justified by the result; the bleeding recurred several times, and in a month the spleen could no longer be felt; the patient had good health for twenty-five years afterwards. Sir Thomas Watson thinks that he has more than once seen the spleen diminish in size in proportion as blood was poured out by the stomach. We may doubt, however, whether in his cases the spleen was primarily diseased, and whether the enlargement of that organ and the hæmatemesis were not joint results of portal congestion, itself due to hepatic disorder.

The two principal organic diseases of the stomach—simple ulcer and cancer—are each very apt to be attended with hæmatemesis. In cases of *cancer,* however, it very rarely happens that any considerable quantity of blood is vomited until the disease has reached an advanced stage, or has, at any rate, declared itself by well-marked symptoms. On the other hand, a *simple ulcer* of the stomach is sometimes latent up to the time when a large hæmorrhage takes place; the patient having either had no symptoms previously, or only such slight indications of gastric disorder as appeared to be of no consequence. This, however, seems to be of decidedly less frequent occurrence than perforation of the peritoneum; which last, as is well known, very often takes place quite unexpectedly in persons who had appeared to be in good health, and whereas perforation is particularly common in young subjects, sudden large hæmorrhage seems to occur rather in persons advanced in years. This kind of hæmatemesis is very seldom immediately fatal. In some cases it returns at intervals of a few hours, and the patient lives five or six days. Sometimes no fresh bleeding occurs for several weeks or even months. The ulcer generally presents certain special characters which will be fully described further on, but Dr Murchison has placed on record two cases, in each of which it was a mere pore-like aperture leading directly into a large branch of artery. A preparation of a similar kind is contained in Guy's Hospital Museum. It is worthy of note that in all these instances the seat of the ulcer was not the lesser curvature, but the cardiac pouch of the stomach. A small ulcer of this kind might easily be overlooked, and it is possible that such may have been the explanation of some of these cases, in which a post-mortem examination has failed to reveal the source of the hæmorrhage. Some time ago a carman, aged thirty-two, who was in the hospital for gout and albuminuria, died suddenly with hæmatemesis. The stomach was full of blood, but I could not discover from what part of the stomach it had come. The vessels of the stomach were not found to be much congested, the hæmorrhage having doubtless emptied them. The liver appeared to be quite healthy. But much more frequently the organ is affected with cirrhosis. Vomiting of blood is indeed very frequent in cases of *cirrhosis of the liver,* and it is often the earliest symptom. Many cases of cirrhosis, in which ascites has already made its appearance, terminate by the sudden supervention of a fatal hæmatemesis.

All systematic writers mention among the varieties of hæmatemesis one in which the effusion of blood is *vicarious* to the menstrual flow. And Sir Thomas Watson relates a case of this kind which came within the knowledge of so able a physician as Dr Latham. A girl about the age of fourteen became the subject of hæmatemesis, recurring at monthly periods. She married without ever having menstruated. She became pregnant; and the hæmatemesis then ceased, and did not return until she had been confined, and had suckled and weaned her infant. Sir Thomas Watson also quotes Mr North as having met with two instances in which suppressed menstruation was followed by repeated and at length fatal hæmorrhage. I have searched in vain for the original record of these cases, so that I cannot tell whether the presence of a gastric ulcer was disproved by an autopsy. All modern clinical observers are, I think, agreed that vicarious hæmatemesis is, to say the least of it, exceedingly rare. And probably many cases which were formerly supposed to be of this kind would now be explained differently.

The occurrence of a profuse hæmatemesis in a person who presents no other well-marked symtoms of disease is then generally the result either of a latent ulcer, or of *simple congestion* of the organ; this last being itself often, but not always, dependent upon latent cirrhosis of the liver. The next question is whether these two conditions can be distinguished from one another. And I think that this must be answered very decidedly in the negative. Both Dr Murchison's cases, for instance, occurred in persons who had been intemperate; one of them was a plethoric woman, aged fifty, the other, a soldier aged twenty-eight, whose liver was cirrhosed. It might well have been thought that in both instances the hæmorrhage was due to mere congestion. So, again, in reading the cases of hæmatemesis which Dr Chambers gives, I find myself quite unable to say what proportion of them are likely to have been cases of gastric ulcer.

Treatment.—The question of diagnosis is one of no little importance, for the practice recommended in cases of hæmatemesis from congestion is hard purging. Sir Thomas Watson directs that five grains of calomel should be given every night and a black dose every morning, till the stools lose their pitchy colour. And he says that he has pursued this plan with perfect success, even when the patient had been blanched by previous hæmorrhages, and when the pulse was feeble and irregular. But it is evident that such treatment must do harm if the blood should have come from an ulcerated artery; and, on the other hand, when the case is one of mere venous congestion, the occurrence of hæmorrhage shows that the vessels are on the way to relieve themselves, even if they have not already done so completely. The administration of astringents at this stage might indeed be injurious. The best course, therefore, is to wait for a few hours, or even for a day or two, until it becomes apparent whether the hæmorrhage is to return. Even if the patient should pass several tarry evacuations in succession, this is probably not to be taken as a proof that bleeding has occurred more than once, for they may all have arisen from a single attack of hæmorrhage. It may therefore be advisable that the patient should take a very gentle laxative, so as to clear out the intestines, and to enable the further progress of the case to be watched more accurately.

But whenever repeated attacks of hæmorrhage occur in a person already blanched by loss of blood, I think it is clear that they depend on something more than portal congestion. Astringents must then be used, of which Dr Wilson Fox considers acetate of lead the best. It may be prescribed in doses of three or four grains, with a quarter of a grain of opium every two or three hours. Some writers have spoken very highly of oil of turpentine, twenty or thirty minims of which are given every four or six hours. Other valuable styptics are gallic acid and dilute sulphuric acid. Of the former ten grains may be administered every two or three hours, or even oftener; of the latter ten or twenty minims. Dr Chambers records a case in which it appeared certain that a gradual oozing of blood, causing continued melæna, was arrested by dilute sulphuric acid, with Battley's solution of opium. The patient may suck small pieces of ice, but he should be allowed to take scarcely anything into his stomach. Starvation is indeed the cardinal point in the treatment of hæmatemesis, nutrient enemata being given if support appears necessary. If the hæmorrhage is profuse the patient's head should of course be kept low. The application of a bladder partially filled with ice on the epigastrium is often serviceable. Dr Brinton recommends that it should be removed when it has been kept on for a few minutes and that it should be reapplied from time to time; but I have not seen any harm arise from its being left in its place for several hours continuously.

GASTRIC ULCER.—In speaking of vomiting, of gastric pain, and of hæmatemesis, I have had to mention ulcer of the stomach as a cause of each of these symptoms. I must now describe it at length.

Ulceration of the gastric mucous membrane may occur under a variety of conditions. I have already mentioned "hæmorrhagic erosions" (p. 121), and both in enteric fever and in diphtheria the surface of the stomach sometimes, though very rarely, presents a number of small ulcers (vol i, p. 283).

In all these diseases, the ulceration of the stomach is due to an acute process, but the organ sometimes presents a large number of chronic ulcers. The most striking instance of this kind that I know of occurred some years ago at Guy's Hospital in a man of colour, aged thirty, who had recently come from the Southern States of North America. He died of thoracic aneurysm, after an illness of twelve months' duration, his principal symptom having been vomiting of food about an hour after its ingestion. Dr Moxon found that almost the whole of the mucous membrane of the stomach was diseased. There were numerous recent ulcers with raised irregular edges, and there were also many thick puckered cicatrices. Such a case, however, is exceedingly rare. It is probable that the disease had commenced on the other side of the Atlantic.

Anatomy.—The affection now to be described differs altogether from this. It is limited to a small part of the surface of the stomach. Most frequently there is only a single ulcer; sometimes there are two; rarely, three, or four, or even five. When there are more than one they generally differ in size and in their other characters, so that it is evident that they have commenced at different times.

One of the most remarkable circumstances connected with gastric ulcers is that their seat is, in the great majority of cases, along the lesser curvature of the stomach. Sometimes an ulcer lies across the curvature itself; more often it is situated either in the anterior or posterior wall, but almost always close to that line. According to Dr Brinton ulcers are found on the posterior surface of the stomach eight times as often as on its anterior surface. But our post-mortem records at Guy's Hospital by no means bear out this statement. Not infrequently two ulcers are found in the same stomach exactly opposite one another, one on each surface. As they generally appear to be of different dates it has been supposed that one of them has been set up secondarily to the other, as the result of its coming into contact with the opposed surface of mucous membrane. Sometimes the pylorus is the seat of the ulcer, sometimes the cardiac pouch. Sometimes, lastly, an affection exactly similar occurs in the first portion of the duodenum; this ought, of course, in strictness to be described among the diseases of the intestines; but it is practically much more convenient to take it with ulcer of the stomach.

An ulcer of the stomach has a sharply defined edge. This at first is entirely free from thickening. Its floor may be formed by the muscular coat, or the ulceration may extend through this, forming a hole, which is always considerably smaller than that in the mucous membrane, and at the bottom of which the peritoneum is visible. Very frequently, unless a different process should be started, the serous coat in its turn becomes attacked; a minute yellow slough forms in it; and the detachment of this allows the contents of the stomach to escape into the general peritoneal cavity, setting up a fatal diffused peritonitis. The ulcer is found in the post-mortem room to have the form of a flattened cone, the base corresponding with the mucous surface of the stomach. It is often described as a "punched-out" ulcer, on account of its regular circular form, and of the evenness of its margin. It is always of small size, being seldom larger than a sixpenny-piece. Not infrequently, instead of eating its way into the peritoneal cavity, it erodes some large vessel in passing through the coats of the stomach. I have already mentioned some cases of this kind in speaking of the causes that may give rise to hæmatemesis.

So far there is nothing in the characters of a gastric ulcer different from those of a similar affection occurring in other parts of the alimentary canal as is sometimes (though rarely) the case. Thus Mr Flower has recorded an instance in which a small round ulcer developed itself in the œsophagus, and passed straight through into the descending aorta. In the duodenum similar ulcers not very infrequently arise after very severe burns of the skin, as Mr Curling many years ago pointed out. And isolated cases have been published in which they have been found in the jejunum, the ileum, and even the descending colon.

But in the great majority of cases, an ulcer of the stomach goes on to acquire further characters which are almost peculiar to it. Its edge, although still perfectly even and regular, becomes slightly thickened, so that it looks as if it were rounded off. For a little distance beyond it, all the coats of the organ are matted together. These changes evidently depend on the occurrence of a chronic inflammatory process; and Dr Wilson Fox describes this as leading to the exudation of a purely granular substance, which contains more or less numerous nuclei, and which ultimately is developed into an imperfectly fibrillated tissue. The peritoneum covering the floor of the ulcer also becomes thickened and opaque and adherent to whatever part may be opposed to it.

Now, I have already remarked that ulcer of the stomach is generally seated in the near neighbourhood of the lesser curvature, and this is particularly the case with the chronic form of the affection. Hence its floor almost always becomes attached either to the under surface of the left lobe of the liver (if it be in the anterior wall of the stomach) or to the pancreas and the adjacent connective tissue and vessels (if it be on the posterior wall). Thus perforation of the serous cavity is prevented, while the ulcer gradually increases in size. Its growth in different directions is not always uniform, and thus it often loses its circular shape and becomes oval or irregular in form. This last, however, is frequently due rather to the circumstance that when two or more ulcers are present they come into contact as they grow larger, and finally coalesce. I have before mentioned that two ulcers are often found just opposite to one another, one on each side of the lesser curvature; these, when they run together, give rise to a single sore, the shape of which may be compared with that of a dumb-bell. The size to which an ulcer of the stomach attains is sometimes very considerable; the 'Pathological Transactions' contain a record of one which measured five and a half by three inches. While thus expanding in circumference gastric ulcers also increase in depth. The peritoneum is gradually worn through where it is adherent, and the surface of the pancreas or of the liver comes to form part of the wall of the stomach. This at first takes place only at a small spot, but the area of adhesion and that of destruction gradually become more extensive. Thus the greater part of the pancreas may in time become exposed in the floor of the ulcer; it is covered only by a thin film of connective tissue, through which its lobulated character can be plainly identified.

In comparatively rare cases, the floor of an ulcer in the anterior wall of the stomach becomes adherent, not to the liver, but to the abdominal walls, and these may in time be perforated, so that a gastro-cutaneous fistula is formed. Dr Murchison has collected twenty-five cases of this kind, of which, however, only twelve were originally instances of simple gastric ulcer; six of them were cases of cancer, seven cases in which the penetration of the walls of the stomach was due to wounds or injuries of the corresponding part of the surface of the abdomen.* A gastro-cutaneous fistula may remain open for several years. It sometimes closes of its own accord.

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* Of these last, the most remarkable of all is perhaps one recorded by Dr Murchison himself of a woman who for three years kept a penny pressed into the sore left by a seton, until an opening into the stomach was formed.

But, as might be expected, this process of adhesion of the floor of the ulcer to different parts is by no means unattended with risk. In the first place the protective process of adhesion may at any time fail to keep pace with the spread of the ulceration, and perforation into the peritoneal cavity may take place, or the adhesions may be broken through in the course of some muscular effort which is made by the patient, when the same result of course follows. For obvious reasons perforation is more apt to occur when the ulcer is in the anterior than in the posterior wall of the stomach; indeed, in the former position ulcers seldom attain any considerable size.

In other cases danger arises from the penetration of blood-vessels. I have already alluded to the fact that the recent "punched-out" ulcer often erodes an artery of some size; but in cases such as I am now describing it is no uncommon thing to see a large artery, or even more than one, with its coats abruptly cut across, lying in the floor of the ulcer, and plugged with a little cylinder of clot that can be pushed out with very little difficulty. In other cases, in which death has been directly due to hæmorrhage, the vessel is patent. The artery itself is sometimes a branch of the coronary artery of the stomach, or the trunk of that vessel, or a pancreatic branch of the splenic artery. Even the trunk of the splenic artery itself is not infrequently penetrated by a gastric ulcer.

Another change to which ulcer of the stomach is liable is its cicatrisation. This occurs not infrequently. Indeed, I think that one scarcely ever sees a large ulcer which has not healed over in some parts of its surface. Dr Brinton speaks of cases in which the whole extent of the ulcer has been found cicatrised, with the single exception of a point in the centre, occupied by an eroded artery, hæmorrhage from which had caused death. But, in most instances, when a gastric ulcer heals, the patient has good health afterwards. Should he die from some other disease the cicatrix varies in appearance according as the coats of the stomach were more or less deeply and widely destroyed; it may show merely a little thickening of the submucous tissue, or it may form a hard puckered mass, with radiating processes extending into the surrounding mucous membrane.

When an ulcer is seated at the pylorus its cicatrisation may give rise to narrowing of that orifice and obstruct the passage of food through it. The result is that the cavity of the stomach becomes dilated and hypertrophied, exactly as in cases in which the pylorus is narrowed by disease of a different kind. Again, the cicatrix of an ulcer occupying the middle of the stomach may, if it was a large one, constrict it, causing what is termed an hour-glass contraction.

Name.—Writers on ulcer of the stomach have assigned different epithets to it, for the purpose of distinguishing it from the more diffused or scattered forms of ulceration, and from cancer. Thus Cruveilhier described it under the name of "simple chronic" ulcer, and Rokitansky under that of "perforating" ulcer. But neither of these names is universally applicable to it. Some cases are not chronic so far as can be ascertained; and in many cases perforation of the coats of the stomach does not occur. I therefore think that one may call the affection "ulcer of the stomach," or "*simple* ulcer" if it be desired to insist on its being distinct from cancer.

Pathology.—The credit of having originally described ulcer of the stomach is commonly assigned to Cruveilhier, who published his account of it in 1830; and after him to Rokitansky, whose work appeared in 1839. But Dr Abercrombie had in reality pointed out all its distinctive characters in 1828. It must, however, be said of Rokitansky that he laid the foundation of the most modern view in regard to this affection, by suggesting that it arose out of a hæmorrhagic erosion. Virchow in 1853 adopted this hypothesis and developed it. He attributed the destruction of the coats of the stomach to the corrosive action of the gastric juice. This, he said, cannot dissolve the mucous membrane, so long as the circulation is maintained, for the alkaline blood will neutralize the acid as it penetrates the tissues. He therefore supposed that the starting-point of the affection was some morbid change in the blood-vessels of that part of the stomach, whether obliteration of an artery, or obstruction of a vein. He also traced the conical form of the ulcer to the distribution of the tuft of vessels arising from a single arterial rootlet. And he discovered a further argument in the fact that, when perforation occurs, the aperture in the serous coat is always to be found in a particular direction, away from the centre of the ulcer. This he attributed to the circumstance that the apex of the vascular cone is likewise excentric, being directed always towards whichever is the nearer of the two curvatures of the stomach, along which the main arterial trunks run. Virchow's hypothesis has since been accepted by many other writers. Panum has endeavoured to support it by artificially injecting little globules of wax into the branches of the abdominal aorta in dogs. He found that when they were introduced into the arteries of the stomach the mucous membrane presented ulcers, which resembled pretty closely, the affection now under consideration in its earlier stage.

It is evident that Virchow's theory consists of two distinct parts. First, there is the question whether ulcer of the stomach at its commencement is caused by arrest of circulation in the corresponding part of the gastric mucous membrane, and begins in a hæmorrhagic erosion. And this must, I think, be decidedly negatived—on the ground that if such were its origin it ought to be frequent in those who suffer from obstruction of the portal circulation, as a result of heart disease or liver disease. Hæmorrhagic erosions are in fact common in cases of this kind, but not, I believe, gastric ulcer. I think therefore that the commencement of the latter affection still remains unexplained.

But a second question is whether the corrosive action of the gastric juice has anything to do with the further development of the affection. That it is not concerned in its commencement is clear, not only for the reason given by Virchow, but also because in its early stage it is exactly like ulcers which may occur in all other parts of the alimentary canal. But, as I have already mentioned, a chronic ulcer of the stomach presents characters which are almost peculiar to it. I believe I am right in saying that they are seen nowhere else, with the single exception of the first part of the duodenum; and this is exposed to the influence of the gastric juice. The only affection with which such an ulcer can be compared is a thick-walled chronic ulcer on the leg. I think it is quite conceivable that the frequent contact of an acid secretion with the surface of an ulcer of the stomach may not only retard its cicatrisation, but also set up a process of chronic inflammation in its edge and floor that may give it its special characters. A further argument in favour of this view may be found in the fact that truncated branches of artery are often exposed in the floor of a gastric ulcer. In all other parts of the body the walls of arteries show a remarkable power of resisting the ulcerative process. Another point requiring explanation is the occurrence of the great majority of ulcers over the lesser curvature of the stomach, or at least in its close neighbourhood; probably, as Sir William Gull suggested, this in some way depends upon the fact that this part is so much more fixed than the rest of the organ; one can easily imagine that its surface may become abraded during the peristaltic movements.

With regard to the *predisposing causes* that favour the development of ulcer of the stomach very little is known. All writers are agreed that it occurs much more often in females than in males; according to Dr Wilson Fox, from twice to three times as often. I have therefore been surprised to find that among thirty-four successive fatal cases at Guy's Hospital, twenty were in men, and only fourteen in women. Eight of the patients were between twenty and twenty-nine years of age, seven between thirty and thirty-nine, one between forty and forty-nine, ten between fifty and fifty-nine, six between sixty and sixty-nine, and two between seventy and seventy-nine. My cases therefore confirm Dr Brinton's statement that there is no special liability to the disease at an early period of life, as has been commonly believed. There is, indeed, one class of cases of gastric ulcer, the subjects of which are particularly often girls at or soon after the age of puberty. I refer to those cases in which the affection remains latent until fatal perforation occurs. Dr Buzzard has even recorded an instance of this kind in a girl nine years old. But it is a great mistake to suppose that the risk of perforation is limited to such cases, and ulcers that have already produced the characteristic symptoms of the disease appear to be equally apt to destroy the patient's life in this way at all ages and in both sexes.

Dr Brinton speaks of poverty and intemperance as other conditions which have an influence in setting up this disease; and, more doubtfully, of fatigue and mental anxiety.

Symptoms.—Of these, the most significant, and generally the earliest, is *pain.* This may be of every possible degree of intensity, from a mere feeling of weight or tightness in the epigastrium, up to the most severe sensations of burning or gnawing or boring, attended with feelings of sickening depression. According to Dr Brinton it is rarely or never described as stabbing or lancinating. In the great majority of cases it comes on in from two to ten minutes after the ingestion of food, and remains an hour or two, subsiding when digestion is accomplished. If vomiting occurs, this almost always brings the pain to an end. Sometimes the pain does not begin until half an hour or an hour after a meal. It is often distinctly increased by the ingestion of hard or indigestible matters, by food which is hot, and (according to Dr Brinton) sometimes particularly by tea and by beer. In very rare cases, on the other hand, it comes on chiefly when the stomach is empty; and it is relieved by food, and even by hot water or by brandy. The pain of gastric ulcer, however, is not always intermittent. It may be continuous, lasting for days or weeks together. These are generally cases in which the ulcer is already of long standing. Whatever its other characters, the seat of the pain is most frequently the epigastrium, immediately below the ensiform cartilage, but sometimes it is described as being behind the cartilage, and sometimes it is three or four inches lower down. Occasionally it is outside the median line, in one or other hypochondriac region. It is usually limited to a very small area, which, according to Dr Brinton, is rarely more than two inches in diameter and is sometimes a mere spot less than half that size.

Scarcely less important than the pain already described, is a pain in the back, to which Cruveilhier first drew attention as a symptom of gastric ulcer. This is of a gnawing character and is generally referred to a single spot between the eighth or ninth dorsal vertebra and the first or second lumbar.

Dr Brinton devoted great pains to the elucidation of the question whether variations in the seat of the pain in different cases could be traced to differences in the position of the ulcer. And he collected some twenty-five cases in which the pain having been referred to one or other hypochondrium, the ulcer was afterwards found to occupy the corresponding extremity of the stomach. He also ascertained that in some cases the position in which the patient chose to lie, as affording the greatest ease to the pain, was a guide to the seat of the ulcer; the prone position indicating that this was on the posterior, and the supine that it was on the anterior wall of the stomach;

whereas, again, when the patient found relief by lying on the right side, the ulcer was at the cardiac end, and when on the left side, it was at the pylorus. But he was obliged to admit that in most cases, whatever the seat of the ulcer, the recumbent posture gave ease, and no information could be elicited as to the effects of changes of position.

Pressure on the spot to which the pain is referred almost always aggravates it, and in many cases there is the most extreme tenderness, so that not the slightest contact with the clothes, nor the gentlest touch from the physician's hand, can be endured. Sometimes pressure on the epigastrium increases the pain in the back. A very few instances have been recorded in which pressure has given relief to the epigastric pain.

Another symptom of gastric ulcer is *vomiting*. In the most marked cases, this does not occur for some weeks after the patient has begun to suffer pain. It takes place when the paroxysm of pain induced by food has reached a certain height; and (as I have already mentioned) as soon as the stomach is emptied the patient is free from all discomfort, or at most experiences a slight burning sensation for two or three minutes longer. The expulsion of the gastric contents is seldom attended with violent retchings. In rare cases, sickness occurs independently of the ingestion of food, but it would appear that this is generally due to some other cause than the ulcer; such, for instance, as alcoholic intemperance.

The vomited matters do not necessarily present any characteristic appearance. But whenever the presence of gastric ulcer is suspected they should always be very carefully examined, and on several different occasions; for proper search may reveal in them the presence of blood. I have already discussed *hæmatemesis* at some length, and pointed out how frequently it is caused by ulcer of the stomach. But what I am now referring to is the presence of blood in small quantities in the vomited matters, so that it fails to attract the patient's notice. As Dr Brinton remarks, it is important that specimens of vomit should be selected for examination which contain as little as possible of the food that has been ingested, and, of course, that they should contain no animal food that had blood-corpuscles in it. If these precautions be taken, he says that a fluid which had seemed comparatively clear will often throw down a sediment containing blood-corpuscles in considerable quantity. In other cases, the amount of blood effused into the stomach is larger. Being altered by the gastric juice, it gives to the vomited matters a brown colour, or (to use the common expression) the appearance of "coffee grounds." It ought perhaps to be noted that the administration, as medicine, of a preparation of iron may give a blackish colour to the contents of the stomach, if the patient should about the same time take tea, or anything else containing tannic acid.

It must not be supposed that the presence of blood in the vomited matters, or even the occurrence of distinct hæmatemesis, is a proof that an artery has been exposed in the floor of the ulcer. The blood often comes either from the minute vessels which supply its surface, or from those which go to the adjacent part of the mucous membrane.

Other symptoms of gastric disorder may occur in cases in which an ulcer is present, but can hardly claim to be regarded as symptoms of this disease. I refer to dyspepsia, flatulence, pyrosis, and constipation. The appetite is generally defective, but in some cases excessive and ravenous, particularly when there is habitual vomiting of all that is taken; sometimes, although there is great desire for food, the patient is afraid to eat, on account of the pain which follows every meal. In young women amenorrhœa attends ulcer of the stomach so frequently that some writers have endeavoured to trace a causal relation between them. The fact, I believe, was first pointed out by Dr Crisp. In older women menstruation often goes on regularly, although they may continue for several years to be affected with gastric ulcer; but if there should be anæmia from profuse hæmatemesis the catamenia may be suppressed for a time.

In long-standing cases there is often extreme wasting; by the constant pain, and the deprivation of food, sharp lines are worn in the patient's face, and cause a peculiar physiognomy. Dr Brinton says that he was often able to recognise the disease at a glance in a crowded hospital out-patient room.

When a gastro-cutaneous fistula has developed itself the patient usually ceases to vomit. Food almost always escapes from the orifice as soon as swallowed; to prevent this the patient has to wear a plug of lint or gutta-percha unless the mucous membrane should happen to protrude, so as to form a kind of natural valve. The general health, however, is often excellent, so that the patient may be able to go about almost as well as before the disease commenced.

The characteristic symptoms of gastric ulcer, then, are the occurrence, soon after the ingestion of food, of a peculiar kind of pain which is relieved by vomiting, or subsides when digestion is completed, and the presence of blood in vomited matters or in the fæcal evacuations. When an ulcer is seated in the duodenum the pain is said to come on between half an hour and two or three hours after meals or even later still; vomiting is not very common, but (according to Krauss, who has published a monograph on this disease) hæmorrhage has occurred in one third of all recorded cases. I may take this opportunity of mentioning that Krauss gives fifty-eight cases of duodenal ulcer in men to six in women.

Latency.—In speaking of hæmatemesis I have already mentioned that ulcer of the stomach is not infrequently altogether latent until it erodes a large artery; and when I come to the subject of peritonitis I shall have to lay still more stress on the same thing, in regard to perforation into the serous cavity. It is partly on this account that I shall postpone, until then, the consideration of the symptoms which indicate the occurrence of this complication. It may indeed sometimes be a question whether the ulcer was absolutely unattended with symptoms during any part of its course, even when a patient who had seemed in perfect health is suddenly attacked with an illness which proves fatal in a few hours. In such cases the ulcer often presents no sign of inflammatory reaction in its walls, and there appears to be no reason why all the coats of the stomach should not be destroyed in a few days, or even in a few hours. Thus Niemeyer relates the case of a young medical man who died rapidly of perforation, and who declared positively that for just a week previously there had been some trifling symptoms which had seemed to him to indicate a slight catarrhal affection of the stomach; and this writer appears to conclude that the time during which the ulcer had been forming must also have been a week. But this conclusion seems to me very doubtful, for in many cases in which perforation occurs in persons supposed to be in good health, the ulcer has smooth rounded edges, and must certainly have existed for a considerable time; and indeed it not infrequently happens that this affection is found accidentally in the body of a person who has had no gastric symptoms, and has actually died of some other disease.

But in other instances, ulcer of the stomach is overlooked, not because the patient has shown no signs of gastric disorder, for such signs may have been observed for several years, but because they have been of so slight a character that the case has been regarded as one of mere dyspepsia. The absence of vomiting appears to be particularly frequent in such instances. Dr Brinton mentions one case in which there was no vomiting in four years during which an ulcer remained active; and he speaks of other cases in which it was represented by slight nausea only, or was limited to a single attack, or occurred only at the very close or the disease. According to this writer, it is upon the size of an ulcer of the stomach that the frequency and intensity of vomiting depend, rather than upon any of its other characters. No absolute rule can be laid down for the diagnosis of gastric ulcer in cases of this kind. Very much depends upon the acumen and judgment of the medical attendant. The existence of the disease may be suspected in many cases, in which the evidence of its presence falls far short of absolute proof. The question of the diagnosis between simple gastric ulcer and cancer will be considered when I come to speak of the latter disease.

Events.—An ulcer of the stomach may have several different terminations. Cicatrisation sometimes occurs, and the patient regains his former state of health. Several years ago some German pathologists at Prague noted the state of the stomach very carefully in a large number of autopsies, and according to their statements both cicatrices and unhealed ulcers were found very frequently, and in the proportion of 147 of the former to 156 of the latter. Scars have sometimes been discovered in the stomach in the post-mortem theatre at Guy's, but very much more rarely. Moreover, since open ulcers are also sometimes found unexpectedly in the dead body we must not lay too much stress on the more or less frequent discovery of cicatrices, as proving that there is much chance of recovery by a patient who suffers from well-marked symptoms of the disease, at least without careful treatment. Certainly I do not myself feel disposed to endorse Cruveilhier's statement that "simple ulcer of the stomach tends essentially to a cure." However, many cases are on record in which symptoms existed and in which cicatrisation has occurred. One such is that of the anatomist Béclard, who suffered from pain in the stomach and vomiting, from which he gradually recovered; when he died, many years afterwards, the scar of an ulcer was found at the lesser curvature of the stomach. Another instance I shall have to mention when speaking of peritonitis. And in the wards I have known several cases in which symptoms of gastric ulcer have been present and in which recovery has taken place.

The healing of a gastric ulcer is not infrequently partial. Cicatrisation may take place on one side of it while in the opposite direction it goes on spreading. Thus the pylorus may become narrowed, or the stomach acquire an "hour-glass contraction" in its centre while part of the surface still remains unhealed, and symptoms of obstruction of the outlet may arise; in addition to these are the proper indications of the presence of the ulcer. Or the sore may heal for a time and afterwards again break out. Probably this is often the cause of the remarkable circumstance that there may be a complete intermission of all the symptoms of gastric ulcer for many months, after which they may again return. In other cases, however, an apparent intermission is really due to the fact that, the first ulcer having finally cicatrised, a fresh one afterwards develops itself. Lastly, it appears probable that the inclusion of nervous filaments in a cicatrix is sometimes the cause of the continuance of pain after the subsidence of the other symptoms; and, indeed, after the cure of the disease.

The *duration* of this disease is often exceedingly protracted. Cases have been recorded in which symptoms were present uninterruptedly for twenty or thirty years or even longer. There are several different ways in which it may prove fatal. Sometimes the patient dies by gradual exhaustion. This, however, seems to occur very seldom, at least I find only three instances of it recorded at Guy's Hospital out of twenty cases in which the immediate cause of death is noted. Dr Brinton speaks of having seen three or four cases of this kind within a few months; but this must be ascribed to the special reputation which he had acquired for the treatment of ulcer of the stomach. On the other hand in eight of the twenty cases death was traceable directly to hæmorrhage, and in nine to perforation. One is therefore compelled to regard these occurrences, not as accidents, but as the ordinary terminations of the disease. Other results of gastric ulcer are comparatively very much more rare. In some cases the destruction of the gastric coats is followed, not by a general peritonitis, but by a circumscribed abscess, which generally occupies the left hypochondrium, and which may in its turn perforate the diaphragm and set up a fatal pleurisy. In other cases again, the disease sets up pylo-phlebitis and abscesses in the liver.

It is a very interesting question whether cancer ever develops itself secondarily in the floor or edge of a simple ulcer of the stomach. Trousseau speaks of the two diseases as antagonistic. But Dr Brinton was disposed to think that the one might pass into the other; and I believe that I have myself seen more than one case in which the stomach presented part of the circumference of a simple ulcer, the rest of which had been replaced by a malignant growth. If this is correct, the occurrence is probably more frequent than might appear from the absence of direct observations of it; for in many cases the extensive development of cancer would doubtless obliterate all traces of the previous ulcer.

Treatment.—There are some cases of ulcer of the stomach in which all that can possibly be hoped for, or all that circumstances permit the physician to attempt, is the palliation and relief of its symptoms. The medicines that are then to be prescribed are those which have already been mentioned at pp. 125, 130, and 135, when I was speaking of gastric pain, of vomiting, and of hæmatemesis respectively. There are, indeed, some further points which I may mention in this place. Thus Dr Brinton said that in certain cases he found a preparation of bismuth, or the *Pulvis kino cum opio* as effectual in arresting hæmorrhage from the stomach as the more powerful astringents. This writer expressed a very strong opinion as to the absolute uselessness of the oxide and the nitrate of silver in this disease; but although it is no doubt impossible that these medicines can act upon the surface of the ulcer as lunar caustic does upon a sore to which it is directly applied, it is yet certain that they sometimes give relief to the pain. On the other hand Dr Brinton spoke very highly of the value of opium, attributing to it, beyond its influence in relieving pain and sickness, a direct power of buoying up the nervous system and supporting the patient's strength. He in fact believed it to be as essential to the cure of an ulcer of the stomach as some surgeons have found it to be in cases of chronic ulcer of the leg. His way of using it was to give a small pill of the watery extract, or a few grains of the compound soap pill, two or three times a day. He prescribed it especially for patients of advanced age, of broken-down constitutions, in whom the disease was of long standing and the ulcer probably of large size.

The use of purgatives requires much caution in cases of ulcer of the stomach, and there is no doubt that they should as far as possible be avoided. Dr Brinton, indeed, speaks of having more than once noticed a definite and repeated coincidence between the occurrence of a paroxysm of pain and vomiting and an accumulation of fæces in the colon; and for such cases he recommends the use of castor-oil. In common with other writers he speaks very strongly against the use of mercury in any form, saying that he is certain that he has witnessed relapses which could only be attributed to its administration.

A blister often relieves the symptoms of gastric ulcer, but it is said that in some patients it causes increased pain. Dr Brinton speaks of having observed this in cases in which there were old adhesions between the stomach and the abdominal walls. This writer thought that leeches should never be used, but Dr Wilson Fox says that the application of two or three of them sometimes gives marked relief to pain. A blister applied to the back has been found to relieve the pain in that region.

But in the great majority of cases something more may be fairly aimed at than the mere relief of symptoms. If there is any truth in the hypothesis that cicatrisation of gastric ulcers is prevented by the action of the gastric juice, the rational treatment is evidently to keep the stomach entirely empty for a time, supporting the patient by enemata alone. In cases of obstruction of the œsophagus life may be maintained in this way for at least two or three weeks; a period which is probably long enough to enable a gastric ulcer to take on a healing action, even if it is not sufficient for its complete cicatrisation. It appears to me that in every case in which the diagnosis is clear, and in which the patient can be induced to submit to it, this method should be carried out for a certain period before any other treatment is thought of. However slight the symptoms may be, one never can tell how near the peritoneum or some large artery may be to the floor of the ulcer; and every week's or month's delay must necessarily add something to its size, and *pro tanto* diminish the prospect of benefit if the patient should at a later period submit to the same plan of treatment.

Again, in many cases, the very urgency of the symptoms affords an additional argument for giving complete rest to the stomach and feeding the patient by nutritive injections. I refer particularly to those cases in which all that is swallowed is shortly afterwards rejected. Vomiting, indeed, as Dr Brinton remarks, is much less amenable to treatment than any other symptom of gastric ulcer, often resisting the action of every reputed remedy. It is also by far the most dangerous and important of these symptoms, on account of the risk that it entails of the supervention of perforation, from the rupture of the protective adhesions. One can therefore never hesitate to recommend that the stomach should be kept perfectly empty for a time, when there is such obstinate vomiting. In some cases, indeed, it may not be absolutely necessary, so far as the mere relief of this symptom is concerned. I have at p. 130 quoted the well-known case of Dr Hunter, in which vomiting that had been uncontrollable was checked by the limitation of the food to milk, given a spoonful at a time. A similar plan was suggested by Cruveilhier, for cases of gastric ulcer. Dr Brinton says that sometimes the milk is better borne when it has previously been boiled, or when it is mixed with lime-water; in some cases he has found a little fresh curd, mixed with a thin pulp of arrowroot boiled in water better than anything else. As convalescence advances, ground rice may be substituted for the arrowroot, and afterwards biscuit powder. Sugar was specially objected to by Cruveilhier, and subsequent writers have endorsed his opinion; it is thought to produce flatulence.

Some persons, however, are unable to digest milk, and according to Dr Wilson Fox there are some elderly people whom it fails to nourish. Animal broths must then be given in its place. But I must add that many patients suppose themselves to be instances of such idiosyncrasies who are afterwards found to do just as well as anyone else upon the most rigidly restricted diet.

During convalescence from ulcer of stomach the most extreme care should be exercised as the patient gradually extends his range of diet. The quantity of food taken at one time must be such as will not distend the stomach; all hot food and drink must be avoided. Complete abstinence from alcoholic stimulants is very important, although Dr Brinton admits that he remembers more than one case in which one or two glasses of sherry, or even one or two tumblers of beer, were taken daily without seeming to impede the cure of what was probably an ulcer of the stomach.

Lastly, it must not be forgotten that pressure upon the epigastrium may do harm when there is a gastric ulcer. A woman should not be allowed to wear her stays, nor a shoemaker to press the last into the pit of his stomach; nor, again, should the physician use any force in manipulating the abdomen. For similar reasons all violent exercise and sudden efforts must be most carefully avoided.

CANCER OF THE STOMACH.—Cancer of the stomach is in every way a disease of great importance. Dr Brinton estimated that it caused 1 per cent. of the total mortality in London Hospital practice, and I find that at Guy's Hospital the proportion is even higher, namely, 79 in 5990 autopsies, or more than 1·3 per cent. According to Dr Brinton it is less common than simple ulcer; but an analysis of the post-mortem records at the hospital during the last twenty years shows that there have been rather more than twice as many cases of cancer as of simple ulcer of the stomach.

Locality.—It is generally said that there are three distinct varieties of this disease, respectively affecting the cardiac orifice, the body of the stomach, and the pyloric orifice. Now, as regards the first of these, I may at once state my opinion that almost all the cases that have been set down as examples of cancer affecting the cardia have really been instances of cancer of the end of the œsophagus extending into the adjacent part of the stomach. So far as I can discover, the 'Pathological Transactions' contain not a single example of cancer beginning in the stomach at its œsophageal end; nor does the museum of Guy's Hospital show any such specimen, in which the lower end of the œsophagus is not also affected. And in every one of the four or five cases that have at different times been recorded under the name of cancer of the cardia in the reports of post-mortem examinations at the hospital during the last twenty years, there is room for supposing that the disease began either in the œsophagus or in the lesser curvature of the stomach. Indeed, on *a priori* grounds we should expect that a part at which the digestive tube is opening out into a large cavity should have little or no tendency to be affected with the disease, in comparison with the narrow passage above it.

Cancer of the middle of the stomach, however, and cancer of the pylorus must be carefully distinguished from one another. And as the latter is the more definite affection of the two, it may conveniently be first described.

The *pylorus*, when affected with cancer, becomes greatly thickened, so that it forms a rounded swelling, which is often somewhat lobulated, and which is almost always sharply defined towards the duodenum, while it passes gradually into the wall of the stomach or extends for some distance along its lesser curvature. The disease generally involves the whole circumference of the orifice, which is consequently much narrowed. It grasps the finger tightly, or may even refuse to admit it; but cases are very rare in which a large catheter cannot be passed into the duodenum. The mucous surface is generally much reddened; it may either be smooth or present nodular excrescences, and sometimes distinct villous growths. Most frequently it is more or less extensively ulcerated.

On making a longitudinal section, one finds that the several coats of the stomach are still plainly to be recognised. The thickest part of the mass is that which corresponds with the submucous connective tissue; this generally makes up two thirds of the whole. Next comes the muscular layer, which is likewise greatly augmented. This appears as a row of pinkish-grey translucent striæ, regularly arranged with opaque bands between them, which last often consist of the cancer tissue. Still further outwards the subserous tissue is also thickened, but to a less extent; and it likewise is generally infiltrated with the new growth. The peritoneal surface may either be unaffected, or it may be the seat of an inflammatory process binding it to the adjacent parts; or, again, it may present more or less numerous cancerous nodules.

Cancer of the *middle of the stomach* is much more variable in its characters. In many cases it begins along the lesser curvature, and then it may either remain limited to that part or spread to one or both surfaces. I have notes of one case of this kind in which only a narrow border along the greater curvature was left untouched by it. In some instances it forms a more or less broad ring completely surrounding the middle of the organ. Sometimes, again, it appears as a large patch, with or without ulceration upon one surface only. Lastly, it sometimes affects the whole of the stomach almost uniformly.

Whatever part of the stomach may be affected with cancer is very liable to become adherent to any other structure with which it happens to be in contact. Most commonly this is the under surface of the liver, and the growth may then extend into that organ, and afterwards it may undergo ulceration; thus a large cavity may be produced, in which ingesta (such as grape skins) may lodge. Or the diseased portion of the stomach may become fixed to the surface of the abdomen. I remember one instance in which the anterior part of the abdominal wall had altogether disappeared, being fused in a mass of cancer two or three inches thick. A gastro-cutaneous fistula may result, but this is rare. Or, again, the growth may become continuous with a congeries of diseased glands near the pancreas, and with that structure itself. Or, perhaps more frequently, the first portion of the duodenum is drawn into adhesion with the back of the diseased pylorus; and sometimes an ulcerated opening forms between them, behind the proper orifice. This depends upon the fact (which I think was originally pointed out by Luschka) that the normal direction of the first part of the duodenum is from before backwards. In other cases the diseased part of the stomach becomes adherent to the colon, and a fistulous communication between them afterwards develops itself.

Histology.—I have hitherto spoken of cancer of the stomach as synonymous with malignant disease of that organ; and it appears that with only rare exceptions this is really the case. Virchow only mentions two instances of primary *sarcoma* of the stomach, one of them being a case recorded by Dr Wilks in the tenth volume of the 'Pathological Transactions.' In each of these cases, and also in a third case of Dr Wickham Legg's (in the twenty-third volume of the 'Pathological Transactions'), the patient was a young girl; and both the ovaries were at the same time affected with sarcoma. The disease seemed to have begun in the lesser curvature. Virchow remarks on the comparatively slight tendency to ulceration as affording a further distinction between this form of disease and carcinoma of the stomach. But in a fourth case of Dr Cayley's, recorded in the twentieth volume of the 'Pathological Transactions,' there were large nodules of the growth projecting into the cavity of the stomach, and these were extensively ulcerated. The patient was a man, aged fifty-seven.

Virchow does not mention any instance in which disease occupying the pylorus was of a sarcomatous nature, but three or four cases of this kind have been observed at Guy's Hospital. One was in a woman, aged forty-seven; the submucous tissue of the pylorus was three quarters of an inch thick. Dr Moxon describes it as thick but flabby, of a milk-white colour, yielding a clear fluid when scraped, consisting mainly of a well-developed fibrous tissue, but also containing some delicate spindle-cells with very large tails. In another case, which occurred in a man, aged sixty-six, the pylorus was the seat of a new growth of yellowish look and of firm consistence, which proved to be a round-cell sarcoma. A third case came under my observation in June, 1876. The patient was a man, aged sixty-seven. The pylorus, through which the finger could readily be passed, presented a large ulcer, seven inches in circumference, the base and sides of which were formed by a homogeneous pinkish-white substance, which yielded no juice, and consisted of round and oval cells, and spindle-cells, embedded in an intercellular substance containing mucin. Probably the cases which Sir Thomas Watson and others have described under the name of simple hypertrophy of the pylorus were also examples of sarcoma. Dr Wilks used formerly to lay considerable stress upon the fact that in some cases of so-called scirrhous pylorus the disease was really only a local thickening of the submucous tissue, with consequent hypertrophy of the muscular coat. He based this opinion partly upon the dry, juiceless character of the growth, consisting mainly of fibrous tissue, partly upon the absence of secondary cancerous nodules. This last fact, however, is not by itself conclusive; for I find that out of forty-one cases of true carcinoma of the pylorus in succession at Guy's Hospital there were at least five in which no cancer existed elsewhere in the body. And, on the other hand, in one case, which would otherwise have been designated simple fibrous thickening or hypertrophy, there were secondary nodules in the liver, the exact nature of which, however, is not specified in the report. One must bear in mind that carcinoma of the stomach (unlike carcinoma of the breast) is situated in what is termed a vital organ. Secondary growths would doubtless be found much more constantly if death did not so quickly ensue.

As I have before stated, in cancer of the stomach the submucous tissue is more thickened than any of the other layers. The older pathologists therefore supposed that the disease began there, but this appears to be a mistake. According to Waldeyer it always commences in an overgrowth of the glands in some one spot in the mucous membrane. These become elongated and dip down into the subjacent connective tissue; when they have reached it they proliferate actively, and so give rise to a cancerous nodule, which spreads out horizontally and may reach a large size, but which is nowhere connected with the superficial glandular layer, except at its starting-point.

Carcinoma of the stomach, as of all other parts, presents in different cases characters which in some respects differ very widely. These in part depend upon the proportion between the amount of fibrous stroma forming the alveoli, and that of the nests of cells contained in them. If the stroma be abundant and the alveoli small, the growth has a tough fibrous appearance and yields but very little juice. If the stroma be scanty and the alveoli large, the growth is soft and of a milk-white colour, giving much juice when scraped. But between the former (which would be termed "scirrhous cancer") and the latter (which would be called "encephaloid or medullary") all gradations exist, and in some cases it may be difficult to say under which head the disease should be placed. As a rule, however, carcinoma of the stomach belongs rather to *scirrhus*. According to Dr Brinton three cases out of four belong to this form of the disease, and among cases affecting the pylorus I believe that the proportion would be still higher. As I have already stated, it passes towards the serous surface between the bundles of the hypertrophied muscular coat, but there may be a partial destruction of these in the centre of the growth. On the other hand, as Dr Moxon has pointed out, *medullary carcinoma* often causes the muscular coat to disappear over a considerable area; so that the whole thickness of the wall is converted into a uniform mass of disease presenting elevations on either surface, but especially towards the cavity of the stomach. Sometimes branching processes sprout from the mucous membrane, which form beautiful microscopical objects, being each made up of a central blood-vessel, clothed with thick layers of well-formed cells. These constitute what has been termed *villous* cancer, which, however, has no claim to be regarded as a separate variety. Dr Moxon has described a case of this kind, in which the floor of the growth was formed by a large mass of soft carcinoma, growing directly into the substance of the liver. The observation is of importance, because it is said that a simple papilloma has sometimes been found in the stomach, analogous to the well-known affection which occurs in the urinary bladder.

In a very few cases a cancerous growth in the stomach has presented the characters of a *cylinder-epithelioma.*

I have already mentioned that the surface of a carcinomatous growth in the stomach is generally ulcerated. Sometimes there are only a few superficial erosions, but very often a deep sore is formed with hard raised ragged edges and a sloughing base. It is possible that the digestive action of the gastric juice may be concerned in bringing about the detachment of large masses of the cancer tissue, which sometimes leads to the erosion of blood-vessels of considerable size. In such cases the ulcer is horribly foul and offensive.

Cancerous growths in the stomach, as in other parts, are liable to undergo *caseous degeneration.* Sometimes scarcely a trace of active growth is discernible; it might be said that the primary affection had undergone spontaneous cure; but the patient has, nevertheless, died of an extension of the disease to other parts.

Another change to which cancerous growths in the stomach are particularly subject is that known as *colloid degeneration.* For a long time pathologists supposed that there was a special form of cancer characterised by its gelatinous appearance. This they termed colloid cancer; but such a view was attended with great difficulties, and not the least satisfactory result of the microscopical researches of the last few years has been the solution of the doubts which prevailed as to the remarkable affection in question. The stomach has always been known to be one of its principal seats. Sometimes the whole thickness of the organ is infiltrated with a jelly-like material, there being nothing to suggest the presence of ordinary carcinoma. But, more frequently, while some parts of the growth have the character of colloid, others have those of scirrhus or of a form intermediate between it and soft cancer. And under the microscope it is not uncommon to find that more or less colloid change is present in cases in which it may not be observed by the naked eye. It is in such cases that the real nature of the affection is most apparent. For it is found that the alveoli are no longer filled with the characteristic epithelioid cells, but that towards their periphery they contain a greater or less quantity of a structureless translucent substance. As this increases the cells become less and less marked and finally disappear. It is said by Rindfleisch that they individually swell out into colloid globes which ultimately blend with the rest of the structureless material that now distends the greatly enlarged alveoli; but he also thinks it probable that a part of this is the result of a chemical change in an albuminous substance secreted from the blood. As the alveoli increase in size they become spherical and the septa between them break down; and thus large translucent globes are formed which under the microscope appear almost structureless. Cancerous growths which undergo extensive colloid degeneration are less apt to infect distant parts than other carcinomata; but on the other hand this form of the affection often spreads over the peritoneal surface, where it produces masses of enormous size. I shall have to refer to it again when I come to speak of diseases of the peritoneum.

To complete my account of the pathological appearances seen in the dead body in cases of cancer of the stomach, I must now refer to certain changes in the form and situation of the organ which arise when the pylorus is the seat of the disease. The obstruction of that orifice may then give rise to an enormous dilatation of the cavity; it may become large enough to hold six or seven pints, and it may fill the whole abdomen, its greater curvature sweeping round just above the pubes. Its walls may at the same time be greatly thickened by diffused hypertrophy of the muscular coat, but sometimes they are exceedingly thin. I shall hereafter have to describe the clinical effects of these changes, but I must add that the occurrence of dilatation is by no means constant. In some cases, probably, cancerous disease of the pylorus fails to obstruct the outflow of the gastric contents; or vomiting is so frequent that no accumulation of them in the cavity of the stomach takes place; or, lastly, the patient's appetite is so bad that scarcely any food is swallowed; while in other cases the extension of the morbid growth along the lesser curvature and into the surfaces of the organ tethers it and prevents its dilatation. Indeed, when the lesser curvature is the original seat of the disease, the cardiac and pyloric orifices may be approximated by the contraction of the growth; and the anterior and posterior walls of the stomach may be flattened against one another so that scarcely any cavity is left.

Of the *causes* of cancer of the stomach very little is known. It occurs chiefly in persons over forty years of age. I find that of forty-six cases, eleven patients only were under the age of 40; of the remaining thirty-five, there were sixteen between 41 and 50, eleven between 51 and 60, and eight between 61 and 70. These numbers correspond generally with those given by Dr Brinton. Among forty-three of my cases, in which the sex was noted, thirty occurred in males, thirteen in females. This also accords with Dr Brinton's estimate; but certain other writers have said that the disease is more common in women than in men. Hereditary predisposition is said to be well marked in some cases. The Napoleon family are cited as affording an instance of this.

In some cases cancer of the stomach has followed a blow upon the epigastrium or some other injury. Andral related an instance in which it occurred in a patient who had taken nitric acid, and Dittrich one in which arsenic had been swallowed. Habitual spirit drinking is a cause which has been mentioned by some writers; and by others the influence of depressing emotions.

Symptoms.—These differ so widely in different cases that it is difficult to give a good description of them. At first they are generally very indefinite; the patient, without apparent cause, begins to complain of a sense of discomfort in the epigastrium after his meals, he has a feeling of weight and pressure there, and he is perhaps troubled with acid eructations. He attributes these symptoms to dyspepsia; but his tongue remains clean, and yet he loses all appetite. Soon he becomes conscious that he is wasting. The uneasiness at the pit of the stomach passes into pain; this may be a dull aching referred to the epigastrium or to the back; or it may be exceedingly severe and of a burning or lancinating character. It is generally more or less increased by meals, but it is by no means limited to the periods at which the stomach contains food. Presently the patient vomits. The times at which he is sick vary with the seat of the disease. When it is the middle of the stomach vomiting may come on soon after meals; when it is the pylorus the food is usually retained for three or four hours until it should be passed on through that orifice. The matters ejected consist at first of partially digested food or mucus, but soon these are streaked with altered blood, which is of a brown or black colour; or they may contain sufficient blood to resemble coffee grounds. Constipation and flatulence are also complained of. The aspect of the patient is altered; he acquires a pale, yellow, waxy look, or his complexion becomes earthy. He is depressed about his condition, irritable, and morose.

These symptoms are by no means conclusive as to the existence of carcinoma of the stomach; they do not warrant more than a suspicion that such disease may be present; a careful physical examination of the patient can alone enable one to speak positively as to the real nature of the case.

But in some instances the greater number of these symptoms are absent. Cancer of the lower end of the œsophagus extending into the adjacent part of the stomach is often latent, and I believe that most of the cases in which carcinoma of the stomach has run its course without producing any marked symptoms have been instances of this kind, such as generally receive the designation of cancer of the cardia. But Sir Thomas Watson relates a very similar case, in which the disease occupied the greater curvature. A gentleman, between forty and fifty years of age, was on his way home from Scotland (where he had been deerstalking and shooting grouse) when he was seized one night in a London hotel with a deadly faintness, very rapid breathing and severe pain referred to the sternum. He had before been gradually losing flesh and strength, but the only definite symptoms of which he had complained were sour eructations, loss of appetite and repugnance to solid food. Sir Thomas Watson could detect no disease; he found the epigastrium full and pulpy. The next night the gentleman had a similar paroxysm and died. The larger curvature of the stomach presented throughout its whole extent a mass of scirrhus, while the cardiac and the pyloric orifices were free.

The examination of the abdomen, when cancer of the stomach is suspected, should be made in a particular way. The patient must be made to lie down, and the front of the abdomen must be exposed. Its shape must then be observed and particularly whether there is any fulness of the epigastrium or of either hypochondriac region, or whether on the contrary they are hollowed. Sometimes a tumour may be seen through the parietes, but most commonly it is to be detected only by manipulation. In some cases it can be felt as soon as the hand is laid upon the surface, but very frequently much care is required. The abdominal muscles are often very rigid, particularly those parts of them which overlie a deep-seated swelling, and careless handling may throw them into contraction, so that nothing can be felt. The patient should be made to draw up his knees and to breathe deeply, making the diaphragm descend freely at each inspiration. If his attention can be concentrated on the inspiratory movements his abdominal muscles will often relax. The broad palm of the physician's hand (which must not be cold) is then to be laid gently upon the part of the abdomen which has to be examined, and it is to be allowed to rise and fall as the patient breathes; gradually slight pressure is to be made, which may be increased until the abdomen has been very thoroughly explored. During all this time the palm of the hand as well as the fingers should be kept evenly applied to the surface; and all sudden movements of the fingers' ends which might excite contraction in the muscles of the abdomen must be avoided. I was taught this method by Sir William Gull. There is sometimes the most extreme tenderness when the stomach is affected with cancer; but even then it is generally possible to make out thoroughly the form and relations of any tumour that may be present, if only due pains and care be taken.

The position and form of the *tumour* produced by cancer of the stomach of course vary greatly in different cases; they are in fact determined by the seat of the growth. If this should occupy the middle of the organ, the left hypochondriac and epigastric regions will contain any mass that can be felt on manipulation. I remember one case in which two nodular ridges could be clearly made out, corresponding one with each curvature; while between them, towards the back of the abdomen, lay an irregular mass which seemed to occupy the posterior wall. In other cases a more or less rounded prominent mass is felt, which is the thickened anterior surface of the stomach. Dr Cayley has related a case in the 'Pathological Transactions' in which the left hypochondrium contained a firm slightly moveable tumour which reached below the umbilicus and was supposed to be the spleen, but which proved to be a stomach indurated by a carcinomatous growth in its walls.

When the pylorus is the seat of the cancer, the tumour is usually much more definite. In some cases its character can be made out almost as plainly during life as when the interior of the abdomen is exposed in the post-mortem room. It forms a rounded mass, often somewhat lobulated, perfectly circumscribed on all sides except towards the left, where it can sometimes be felt to pass gradually into the wall of the stomach; it may vary in size from that of a walnut to that of a Tangerine orange. Its seat is usually a little above and to the right of the umbilicus; considerably lower than the position of the healthy pylorus, which, indeed, lies so completely under cover of the liver as to the inaccessible to palpation. By Dr Brinton, the fact that a cancerous pylorus is felt in the umbilical region seems to have been recognised only in the case of female patients, and he attributed it to the alteration in the position of the viscera caused by the use of stays; but this explanation is clearly insufficient, since the same thing is frequently observed in males. It probably results from the traction exerted upon the lesser omentum by the weight of the tumour. Thus, when that fold of peritoneum is thickened and involved in the growth, or when the pylorus is retained in its normal position by adhesions, no tumour can be discovered. On the other hand it sometimes descends very much lower. Dr Wilson Fox says that it may be found in the right iliac fossa or even in the pelvis, adhering to the intestine, uterus, ovary, or bladder. I have notes of one case in which a tumour in the *left* hypochondrium, whose exact situation varied at different times according as the stomach was more or less distended, proved to be the pylorus, which had been dragged over to the left side, and was firmly adherent to the parietes and to the edge of the liver. A scirrhous pylorus usually seems to move slightly downwards when the patient draws a deep breath; perhaps the liver, having itself to descend, pushes it down in its turn. Some observers have thought that the movement is rather apparent than real, the truth being that the expansion of the ribs carries the abdominal walls upwards over the tumour; but I feel satisfied that this explanation does not meet the facts.

The tumour caused by cancer of the pylorus often receives an impulse from the abdominal aorta.

It is said sometimes to disappear entirely for many days together, this being due either to twisting of the stomach on its axis or to its being overridden by a distended colon. To percussion it should yield a dull note, but when it is of small size this may be masked by the resonance due to the adjacent intestine. I also remember one case in which the tumour formed (so to speak) the roof of a hard-walled ulcer communicating freely with the cavity of the stomach, and in which it distinctly gave a very resonant sound when percussed.

But the results of manipulation of the abdomen in cases of cancer of the pylorus are by no means limited to the discovery of a tumour; one has also to ascertain, if possible, the position and size of the stomach itself. I have already remarked that this organ is often greatly dilated, and descends much lower in the abdomen than under normal conditions; the greater curvature may be below the umbilicus, and may even reach down to the pubes. The organ then resembles a large flaccid bag; and the epigastrium and left hypochondriac regions are deeply hollowed, while the lower part of the abdomen is protuberant. This, indeed, is not by itself proof of dilatation of the stomach, for it is common enough in persons whose small intestines are distended with flatus, if their abdominal walls are also loose and flaccid. What is conclusive is the detection of the peristaltic movements of the thickened organ. If the surface of the abdomen be attentively watched, a wave of contraction may often be seen to start from the left hypochondrium, descend below the umbilicus, and pass on to the right side, and then a little upwards towards the cartilages of the right ribs. Or a rounded protuberance, as large as an orange, may rise up on the left side and travel round to the right in the same way as the wave. In one or two cases I have seen distinct antiperistaltic movements (from right to left) in a hypertrophied stomach. Another indication of enlargement of the stomach is the production of a splashing sound by manipulation of the lower part of the abdomen; but this is not a sign of much value by itself, for similar sounds may be produced from the presence of gas and fluid together in coils of the small intestine. If there should be occasion to pass an œsophageal tube down into the stomach, the end of it may sometimes be felt through the abdominal walls. According to Leube, it may, in health, reach as low as the umbilicus; but if it descends below the level of the *crista ilii* the stomach must be dilated. He lays great stress on this in diagnosis.

It will be observed that the physical signs of this form of chronic dilatation of the stomach differ in some respects from those of the acute paralytic distension of the organ which I described at p. 117.

The presence of dilatation of the stomach modifies to a considerable extent some of the other symptoms. I have already mentioned that in ordinary cases of cancer of the pylorus the patient vomits about three or four hours after each meal, at the time when digestion is completed, and when the food ought to be passing on into the duodenum. But when the cavity of the stomach is enlarged, three or four meals may be retained in succession; and the patient, when he does vomit, may bring up surprising quantities of fluid, several pints at a time. I remember one instance in which vomiting never occurred except at night; and in some cases the stomach may reject its contents only at intervals of some days. The matters vomited in cases in which the stomach is dilated also present characters different in some respects from those which are observed under other conditions. They generally consist of a thin highly acid fluid, of a dirty grey, brownish, or greenish colour, which, on standing, becomes covered with a thick frothy yeast-like scum, while it also deposits at the bottom of the vessel a more or less abundant sediment. In the scum, as well as in the fluid, oval spores and beaded tubes of the *Torula cerevisiæ* (or yeast plant) are often found in large quantity; and also in most cases certain little flat rectangular bodies, which most observers believe to belong to another microscopic plant. They are often present in enormous numbers, and have the remarkable peculiarity that they are divided by cross lines into smaller rectangles, some of them into four, others into sixteen, and some even into sixty-four, according to their size. They thus resemble packages tied across again and again by cords; and Goodsir, who in 1842 was the first to observe them, gave them the appropriate name of *Sarcinæ ventriculi* (*sarcina* = woolpack). Dr Chambers has pointed out that they may be found in the dead body in the stringy masses adherent to the interior of the stomach; and therefore he regards this as the appropriate seat for them, and not the liquid which the organ may happen to contain.

Another circumstance that may considerably modify the symptoms of cancer of the stomach is the formation of a gastro-colic fistula. The best account of this affection is that which Dr Murchison gave in the 'Edinburgh Medical Journal' for 1857. In almost all such cases the patient vomits fæcal matter; it has been pointed out by Dr Gairdner that the only exceptions to this rule are cases in which there is at the same time narrowing of the pylorus, so that the stomach is constantly kept overloaded with its proper contents. The patient's breath often has a fæcal odour, or he has eructations of intolerable fœtor, or a horrible taste of fæces in his mouth. Much less frequently the effect of the fistula is to allow matters to pass from the stomach into the colon; at any rate, there are only seven out of twenty-three cases collected by Dr Murchison in which undigested matters were recognised in the fæces. This constitutes what is termed *lientery*. It must not be supposed to be of itself a proof that a fistula exists. The attempt has been made to increase its significance by giving food coloured with cochineal to patients who pass undigested matters from the bowels; and by observing what length of time elapses before the colouring matter appears in the evacuations. In a case of Schönlein's this occurred only at the end of twelve hours, in the last of seven evacuations that took place during the interval. He inferred that the case was not one of gastro-colic fistula, and (with less reason) that the lientery was due to widening of the pylorus. In patients who have an opening between the stomach and colon the appetite is generally very bad, but in one instance there was craving for food. Pain is not invariably present. Indeed, the formation of the fistula sometimes leads to the relief of pain that had before existed.

Returning now to the consideration of the symptoms of cancer of the stomach in general, I have to add that towards the fatal termination of the disease they often subside to a very great extent. I have notes of one case in which vomiting did not occur once during the last month of the patient's illness. Fresh symptoms, however, often arise; ascites, from extension of the cancerous growth to the peritoneum, or from compression of the portal vein; jaundice, from interference with the common bile-duct; œdema of one or both of the lower limbs, from thrombosis of the corresponding femoral or external iliac vein or veins. In some cases the patient lies for several days before his death with cold blue extremities and with a scarcely perceptible pulse, but suffering no pain.

Prognosis.—The duration of cancer of the stomach cannot be stated with precision, because we have no means of fixing the date of its commencement. But it seldom fails to destroy life within a short period from the time of the first appearance of well-marked symptoms. Dr Brinton estimated this period as amounting on an average to twelve and a half months. Niemeyer says that the disease generally proves fatal in from five to fifteen months. Dr Wilson Fox says that the most rapid case he can find recorded is one by Valleix, in which death occurred in four months. But at Guy's Hospital three cases have occurred, in which the duration of the symptoms was stated at one month, eight or nine weeks, and three months respectively. The longest case mentioned by Dr Fox was one in which the patient lived three and a half years after the appearance of the first distinctive symptom; but he cites from Abercrombie the case of Napoleon, who had paroxysms of severe pain for nine years before his death in St Helena. One remarkable case is recorded at Guy's Hospital in which the patient had suffered for seven years from symptoms of disease of the stomach.

Diagnosis.—That the detection of cancer of the stomach is often a very difficult matter must be sufficiently evident from what I have already stated. There are, indeed, some cases in which the most acute observer cannot do more than suspect the presence of the disease. The only rule which I can lay down is that whenever symptoms of dyspepsia come on without apparent cause in a patient over forty or fifty years of age, and are accompanied with great and rapid loss of strength and of flesh, the possibility that cancer of the stomach may be developing must always be borne in mind.

Even when the symptoms point clearly to the existence of serious organic disease of the stomach, there always remains the question whether this disease is simple chronic ulcer or cancer. Between these affections the diagnosis is often perfectly easy. In very young persons, malignant disease of the stomach may be dismissed from consideration. It scarcely ever occurs except in the form of a sarcomatous growth, generally affecting also the ovaries, and attended with ascites, while it produces comparatively slight gastric symptoms. And in older individuals there are many points of distinction. In cases of ulcer, the pain and sickness bear a much closer relation to the time at which food is taken than in those of cancer. Vomiting of blood in considerable quantity is much more apt to occur at an early stage of the disease, and life is prolonged to a much later period. Any case in which well-marked symptoms have existed eighteen months or longer may generally be pronounced to be one of simple ulcer and not of malignant disease.

On the other hand, cancer of the stomach may for the most part be diagnosed whenever a tumour is discovered having the characteristics above described. Cases of simple ulcer affecting the pylorus have indeed been placed on record in which this part has been so thickened and indurated that the presence of a scirrhous mass has been simulated. And when an ulcer occurs at this part of the stomach it tends, when it heals, to narrow the orifice, and so may give rise to further symptoms resembling those which result from cancer. Dr Brinton says that he has met with one or two cases in which during the whole progress of the disease there was nothing to justify a positive diagnosis.

In considering the diagnostic value of pyloric tumour, I must further mention that it is sometimes present when there are no other symptoms of gastric disease. Sir Thomas Watson relates a case in point. A young woman had a pulsating tumour in the epigastrium which was at first supposed to be an aneurysm, and afterwards a mass of fæces in the colon. She had no sickness nor any gastric symptom. The tumour proved to be a cancerous growth in the stomach; it lay in front of the abdominal aorta.

On the other hand it must not be forgotten that in many cases of cancer of the stomach and even in some cases of cancer of the pylorus, no tumour is at any time to be discovered.

With regard to the *treatment* of cancer of the stomach there is very little to be said. It is important that the patient's strength should be saved as much as possible; and on this account the range of food should not be too strictly limited. The patient must not be subjected to the starvation plan of treatment recommended for cases of simple gastric ulcer. Alcoholic stimulants may be allowed in small quantities.

Medicines are required only for the relief of particular symptoms. The remedies available in the treatment of gastric pain, of vomiting, and of hæmorrhage, have already been fully discussed at pp. 126-7, 130, 135 respectively.

There is, however, another symptom, of the management of which nothing has as yet been said. I refer to the vomiting of liquid which forms a yeast-like scum and which contains sarcinæ. For it Sir Thomas Watson recommends common salt, creosote, and the sulphites or hyposulphites. The last-named remedies were first suggested by Sir William Jenner. The dose of them is stated at from fifteen to thirty grains, which should be taken soon after meals.

But the presence of sarcinæ in the vomited fluids is in most cases an indication that the stomach is in a state of chronic dilatation, and this is a condition about which there is still something to be said. I have already mentioned it as an effect of cancerous disease of the pylorus, and have described the physical characters by which it may be recognised; and I have remarked that it may also follow the cicatrisation of a simple ulcer affecting the pyloric region. Those writers who admit that the pylorus is liable to a simple hypertrophy regard this as another occasional cause of dilatation of the stomach. Paralysis of the muscular coat limited to the pyloric portion is also mentioned by Dr Wilson Fox, who quotes two cases from Andral in which the stomach was greatly enlarged without there being any actual narrowing of the pyloric orifice. But in one of these instances there was extensive ulceration of the pylorus; and in the other its walls were indurated, although the muscular coat was atrophied. And even in cases of cancer affecting the pyloric orifice, it not rarely happens that after the ordinary symptoms have developed themselves and advanced to a fatal termination it is found on post-mortem examination that the finger can be readily passed into the duodenum. Evidently, then, the outflow of the contents of the stomach may be arrested, without there being an actual closure of the channel.

Still, on the other hand, I think it is doubtful whether this kind of chronic dilatation of the stomach ever occurs independently of obstruction of some kind at the pyloric orifice. Writers mention hysteria, hypochondriasis, and diseases of the nervous centres as possible causes. Hodgkin pointed out that the organ is often very large in Indians, who eat enormous quantities of vegetable food. Bamberger says that it may be due to dragging down of the stomach by an omentum adherent to a hernial sac.

The exact determination of the various possible causes of chronic dilatation of the stomach is becoming a matter of considerable importance, in consequence of the success which has recently been attained in cases of this kind by the systematic use of the stomach-pump. This treatment was first proposed by Prof. Kussmaul, of Freiburg [now of Strassburg], in 1868. A tube should be introduced into the stomach every day. As much of the contents as will come away readily should first be withdrawn. Some tepid water is then to be injected and afterwards to be pumped out again, and the process should be repeated two or three times until what returns is almost clear. Weak solutions of carbonate of soda, or of permanganate of potass, or even of creosote, may also be thrown into the stomach in some cases. This plan has been tried in a few instances at Guy's hospital and with decided success. And Dr Schliep has recorded some other cases of the same kind in the Clinical Society's 'Transactions' (vol. vi, p. 41). The first introduction of the tube is exceedingly disagreeable to the patient; but before long he becomes accustomed to it; and he is even glad to pass it himself, so great is the relief which he experiences from its use. The vomiting often ceases entirely; there may be great diminution of pain; the appetite improves considerably, the patient becomes much more cheerful; he regains much of the flesh and strength that he had lost, and he is no longer troubled with constipation.

Such striking results are not, however, to be looked for in those cases in which dilatation of the stomach is the result of cancerous disease of the pylorus.

Gastric induration.—Another organic disease of the stomach, to which I can only refer very cursorily, is one in which its walls are uniformly thickened, without the development of any morbid growth, its cavity being at the same time greatly reduced in size. This affection is spoken of by systematic writers as "cirrhosis" or "fibroid induration" of the stomach. I shall hereafter have to mention it as being occasionally the starting-point of a general chronic peritonitis; but I am not sure that it always sets up such an affection. The coats of the organ may be from half an inch to an inch and a half thick, and only capable of receiving four or five ounces of fluid. The symptoms of this affection are exceedingly obscure. A tumour may be discoverable, and this may be more or less resonant on percussion. Probably it is impossible to distinguish cases of this kind from those of diffused sarcoma of the stomach, to which I have already alluded when speaking of cancer.

This fibroid thickening of the coats of the stomach has probably its first stage in the thick, pigmented, almost warty condition (*état mamellonné*) of chronic gastritis. It chiefly affects the pyloric region and occasionally forms what may be termed a non-malignant fibrous tumour of the pylorus. Sometimes, however, it extends to the whole of the stomach which, to use Dr Bristowe's words, "retains its form like an india-rubber bottle."

Gastric concretions.—Lastly, brief reference must be made to certain very rare cases in which immense masses of hair and string, matted together and moulded to the shape of the stomach, have been found in its cavity, and in that of the upper part of the intestine. Sir William Gull brought a case of this kind before the notice of the Clinical Society in 1871, and another was related at a meeting of the Pathological Society by Mr Pollock. In the former case the mass, when dried, weighed five and three quarter ounces; it was composed of string, thread, cotton, wool, and hair of three colours, that of the patient herself (aged thirty-two) and of her children. She had never been noticed to eat hair; but the person from whom Mr Pollock's specimen was taken, and who was a delicate girl aged eighteen, had been observed to put hairs into her mouth when only three or four years of age. In that case a projecting tumour, the size of a large orange, was felt in the epigastric region during life; it was apparently solid and very slightly moveable. A tumour was also felt in a third case, referred to by Sir William Gull. It occurred in a woman aged thirty, who for fifteen years had indulged in the habit of eating her hair, and who had suffered all the time from pain in the stomach, but had worked as a servant until six years before her death. In that case the mass weighed thirty ounces. None of these patients were of unsound mind; in lunatics I believe a similar condition is not infrequent. A fatal termination appears generally to occur sooner or later from perforation of the stomach, with consequent acute peritonitis.

DISEASES OF THE INTESTINES

Colic.—*Onset—Causes—Diagnosis—Prognosis and treatment.* Lead-colic.—*History of its recognition—Modes of infection—Diagnostic characters—The blue line—Pathology and treatment.*

Constipation.—*Origin—Effects—Treatment—Intestinal casts.*

Diarrhœa.—*Acute and epidemic form—Chronic form—Tubercular enteritis—Diarrhœa from lardaceous and malignant disease—Symptoms—Treatment.*

Typhlitis.—*Relation to perityphlitis and disease of the appendix—Diagnosis and treatment.*

Dysentery.—*Anatomy—Catarrhal and diphtheritic forms—Sporadic and epidemic dysentery—Ætiology—Symptoms, diagnosis, course and event—Treatment.*

Intestinal Obstruction.—*Invagination—Impaction—Stricture and contraction—Strangulation and volvulus—General morbid anatomy and symptoms—Special symptoms in chronic obstruction—Its treatment—Symptoms and diagnosis in acute obstruction—Treatment.*

Intestinal Entozoa.—*Tapeworms—Round-worms—Thread-worms—Sclerostoma—Trichinæ—Preventive and curative treatment.*

That part of the alimentary canal which lies below the stomach is liable, like almost all other organs of the body, both to disorders of the function and to diseases of structure. As usual, we will begin with the former.

Colic.—*Enteralgia.*—Of this malady the main symptom is pain, of a twisting or dragging or wringing character, generally referred to the umbilicus or to some spot in the upper part of the abdomen. It comes on in paroxysms which are often of extreme severity; but during the intervals the patient may be perfectly easy, and there is frequently no tenderness on pressure. The patient rolls about in the hope of finding relief, or lies on his stomach with his hands clasped together beneath him, or leans with the whole weight of his body across the back of a chair. In some cases, however, pressure increases the pain, especially when parts of the intestines are distended with gas.

An attack of colic is often attended with nausea or vomiting. Writers say that the skin is cool, and that the pulse is often slower than natural. But I remember a case of severe abdominal pain, admitted into hospital one night, in which the fact that the temperature was two or three degrees higher than normal led to some doubt as to its real nature, but next morning the patient was well, and a review of the symptoms seemed to prove that the attack had been one of colic. In another case that I lately watched for some hours with not a little uneasiness, the skin was covered with a profuse cold sweat, and the pulse was much quickened. The expression was anxious, but there was not the peculiar sunken look of the features which belongs to the more dangerous forms of abdominal disease.

The *immediate cause* of colic appears to be a spasmodic contraction of some part of the large (or perhaps of the small) intestine. Associated with this there may be an accumulation of gas in adjacent parts of the bowel;

and the attacks of spasm are then attended with rumbling noises, "borborygmi," which are audible to the patient and those about them. In such cases there may be partial distension of the abdomen; but in typical colic it is hollow and retracted, its parietes are hard, and the muscles feel as if drawn up into knots.

A very important cause of colic, and one that must never be forgotten, is the presence of lead; this form will be described separately.

In some cases colic is due to the ingestion of food which irritates the alimentary canal. Perhaps the most marked instances of this kind are those in which persons are attacked by it after eating meat or game which is "high." Unripe food, mushrooms, ices are also mentioned as being capable of exciting it. Another cause is commonly said to be exposure to cold, but of this I am by no means sure. The majority of purgative medicines give rise to intestinal pains which are essentially the same as those which characterise colic. Under some of these conditions the complaint is associated with diarrhœa, or the patient's sufferings may go on increasing in severity until one or more loose evacuations are passed, whereupon the pain ceases for a time or even altogether. But in the more typical cases of colic constipation is a prominent symptom, the attack has no tendency to terminate of its own accord by the bowels acting, and the administration of medicine is necessary to bring about this result. In such instances the exciting cause is generally the presence of hard scybalous masses in some part of the large intestine; these can often be plainly felt through the abdominal walls.

From what has been said in the previous paragraph it must be evident that colic cannot be regarded as a true neuralgia in the sense in which that term is used in the present work; and accordingly Anstie speaks of it as being quite independent of the operation of the neurotic temperament.

The *diagnosis* of colic is often not difficult, but frequently needs the utmost caution. Some of the most dangerous forms of inflammation to which the abdominal viscera are liable may for the first few hours present very similar symptoms, and a mistake may be attended with fatal consequences. Certain writers, indeed, define colic merely as a painful affection, dependent on spasm of the bowels, and they say that it is really present in cases of acute peritonitis, and the like, but that it then constitutes a minor feature of the disease. But I think that the more convenient plan is to limit the use of the term to those instances in which the pain is, so far as our science carries us, the substantive complaint. And the rule must then be that no case should be set down as one of colic, and treated so, unless its characters are such as conclusively to prove that no serious mischief is present. The most important of these are a retracted, hard, knotted state of the abdomen, the fact that pressure relieves the pain, and the absence of anxiety of countenance and of constitutional disturbance. Sometimes important light is thrown on a case by the fact that the patient has had former attacks of exactly the same kind, which have passed off entirely within a few hours; or, again, by the confession that he has not long before eaten some particular article which he knows by previous experience to be capable of powerfully irritating his large intestine.

But colic has also to be distinguished clinically from some other affections which are no more grave than itself. One of them is a form of gastric pain which has already been described at p. 125. As in that complaint the epigastrium is prominent, colic can be confounded with it only when the bowel is considerably distended with flatus. The real nature of the case, however, can generally be made out by gentle percussion; the note being (as Dr Wilson Fox observes) less prolonged and higher pitched over the colon than over the stomach. Moreover, the pain has seldom exactly the same position in colic and in gastrodynia; in the former it often extends into the right hypochondrium or downwards into the left iliac fossa in the direction of the sigmoid flexure, whereas in the latter it is absent from these regions of the abdomen. The so-called "biliary colic" and "renal colic" have also to be distinguished from the ordinary intestinal colic; but I think it must be admitted that there may be cases—particularly those which female patients commonly call "spasms"—of which it is very difficult to say that they are more likely to belong to one than to another of these affections.

An attack of colic always terminates in the recovery of the patient, and that within a few hours, or a day or two at the outside. It is therefore of comparatively little importance whether or not active treatment be adopted, particularly at the first onset. And since some of the drugs by which this complaint would be shortened are precisely those which would do the greatest possible harm if the disease should be commencing peritonitis, one cannot be too careful to avoid interfering in any case as to the nature of which it is possible that doubt can be entertained.

Colic, in fact, affords the only exceptions to the rule which I have heard Dr Wilks lay down, that whenever a pain in the abdomen is so severe as to cause the patient to send for a medical man, this *ipso facto* proves that the administration of a purgative is unjustifiable. The rule itself is of great value and should be kept constantly before one's mind. But I feel sure that there are at least some cases in which it ought to be infringed. The patient may clearly owe his attack to something which he has eaten and which has disagreed with him; he may have suffered in the same way before, and have quickly got well after taking a purgative; his abdomen may be retracted and hard, the pain may be relieved by pressure, and it may be entirely paroxysmal, with complete intermissions. Under such circumstances one is fully justified in giving him at once an ounce of castor-oil with twenty or thirty minims of tincture of opium, and in directing that half as much should be taken again at the end of three hours if the bowels should not have acted in the meantime. Enemata of turpentine or assafœtida may be prescribed if there should be any accumulation of gas in the intestine. The abdomen may be rubbed with a stimulating embrocation, or a tin filled with hot water may be laid across it, or a cloth folded round a quantity of hot bran.

LEAD-COLIC.—In speaking of the causes of colic I reserved for separate description a form of it due to the absorption of lead into the blood.

Long before this fact was ascertained, the complaint itself was well known as of endemic occurrence in certain parts of England and of the Continent. Thus it prevailed in Poitou, and was called *Colica pictonum;* in Devonshire, so that within the five years ending in 1767 two hundred and eighty-five cases of it were said to have been admitted into the Devon and Exeter Hospital; and in the West Indies, where it received the very appropriate name of the "dry belly-ache." In each instance it was at an early date attributed to some beverage which the people habitually drank; in Poitou to wine; in Devonshire to cider; in the West Indies to rum. In the first half of the eighteenth century Huxham endeavoured to refer it more definitely to the "tartar" contained in each of these drinks.

The discovery that this form of colic is due to the action of lead was first made by Sir George Baker, whose paper on the subject, read at the College of Physicians, in 1767, is still quoted as a masterpiece of inductive logic. He showed that in the counties of Hereford, Gloucester, and Worcester persons who drank cider did not suffer from colic; and then that Devonshire cider contained lead, whereas Hereford cider was free from it. Next he traced the presence of the metal in the former to the circumstance that lead was used in the construction of the cider-presses, and that leaden weights were sometimes put into the casks to prevent its getting sour. Not long afterwards it was shown that preparations of lead were added to the wines made in Poitou with the very same object, that of neutralising acidity, and that in the West Indies the stills in which rum was made had leaden worms, which impregnated it with the metal. I must not omit to mention that one important link in the chain of evidence connecting these facts together, was the circumstance that both in Poitou and in Devonshire a peculiar form of paralysis affecting the upper limbs was commonly associated with the colic. This also was at the same time traced to the poison; I have described it in the first volume of this work (p. 447).

The recognition of the cause of the complaint of course led to the disuse of those particular methods which had been concerned in introducing the metal into the human body, and endemic lead-colic soon became a thing of the past. But the affection itself is still often met with. Thus persons engaged in the manufacture of white lead are very apt to be attacked by colic. This is believed to be mainly due to the diffusion of the carbonate in a pulverulent form throughout the workshops, so that, besides being inhaled in respiration, it collects upon the hands and is carried into the mouth with the food. It is doubtful whether lead is ever absorbed directly through the skin. Painters and plumbers often suffer from the disease. It is sometimes observed also in glassmakers, enamellers, shotmakers, printers, and typefounders, but not so commonly as was at one time supposed. In all these occupations it is said to occur more seldom than formerly, and if the men were but sufficiently careful it is probable that only those employed in making white lead, and perhaps those who make sugar of lead, would ever be attacked with lead-colic.

The directions given to workmen whose occupations bring them into contact with lead are chiefly that they should pay great attention to personal cleanliness, and that they should be very careful not to let the poison enter their air-passages, and above all not to swallow any particles with their food. They should have an outer suit of linen clothing, worn only while they are at work, and washed at least once a week. They should never take their meals in the workroom. When there is much dust, masks or respirators would probably be useful, but the men can seldom be induced to wear them. Some years ago Liebig recommended the habitual use of "sulphuric acid lemonade,"—a liquid containing a small quantity of sulphuric acid sweetened with sugar which it was supposed would render any compounds of lead that might enter the stomach innocuous, by converting them into an insoluble sulphate. And Sir Thomas Watson states that in some works at Birmingham the addition of the acid in question to the treacle beer which the men drank caused the disappearance of colic from among them. It had before prevailed to a distressing extent; afterwards not a single case occurred for fifteen months.

In other cases, however, the complaint results from the impregnation of drinking-water, or of some article of diet, with the poison, and this source of the disease is the more important because it is especially liable to be overlooked. A well-known instance is that of the family of King Louis Philippe when living in exile at Claremont: several persons were attacked at the same time. The amount of lead in the water which they drank was seven tenths of a grain per gallon. Now, it is well known that water containing carbonic acid and certain salts of lime has less action on metallic lead than water which contains such ingredients only in minute proportions or from which they are altogether absent. Thus the distilled water sometimes used for drinking purposes on board ship is particularly liable to be impregnated with the metal; and even zinc vessels may contain enough lead to make distilled water which has stood in them injurious to health. I must add, however, that the cause of colic occurring on board ship, especially in the French navy, has been a matter of much discussion; certain writers are still of opinion that it may be due to some climatic condition or dietetic cause of which the nature is as yet unknown.

As an example of the production of colic by lead contained in food, I may mention the case (quoted by Sir Thomas Watson) of the troops at a station in Ceylon in 1832. More than seven tenths of those who made up the force were attacked, and the cause was found to be the presence of this poison in some coarse sugar which had been distributed among the soldiers from one particular estate. Lead-poisoning, too, has been sometimes known to arise from the use of snuff with which the red oxide, or the yellow chromate, had been mixed, or which had been fastened up in metallic foil made of this metal.

Ordinary colic and the affection caused by lead do not differ in their symptoms. There is, however, this peculiarity, that whereas mild cases of the former scarcely ever occur, at least so as to come under medical observation, there are not a few instances of the latter in which the pain is of trifling severity and the abdomen soft and supple. Thus the patient may continue to suffer from the complaint for a considerable period, and yet go on with his work, absorbing all the time more and more of the poison.

What alone enables one to speak positively as to the real nature of such cases is the fact that the presence of lead in the body is revealed by a peculiar discolouration of the gums. This was first pointed out by Dr Burton in 1840, and it is commonly spoken of as the "blue line." The name, however, is unfortunate, and has no doubt often led to mistakes; for under conditions of irritation the margin of the gums is very apt to present a bluish-purple border, which has nothing whatever to do with the presence of lead. The line which is really characteristic presents peculiarities which I believe were first noticed by Sir William Gull. It consists at first of a single row of black dots, corresponding with vascular papillæ in the normal mucous membrane. Mr Tomes several years ago explained its formation, proving that it was caused by a chemical action between the lead and the tartar round the teeth. And at the same time he pointed out that in all probability the constituents of the tartar itself are not concerned in its production, but rather animal matters which had penetrated into the pores of the tartar, and the decomposition of which would give off sulphuretted hydrogen. He showed that where there is a gap between the teeth, and no tartar, no "blue line" is formed. Further evidence of the same fact is afforded by cases, of which I have seen more than one, in which persons who have kept the teeth very clean have failed to present the line, although they were indubitably affected by lead. In many cases, too, the line is exceedingly partial, even when it exists. There may be only two or three black dots on one or more of the processes of gum projecting up between the teeth at the side of the mouth; and a lens may be necessary to enable their true nature to be determined. Some writers have supposed that the lead with which the sulphuretted hydrogen combines was previously in organic combination with the elements of the tissues. But more probably it really comes directly from the circulating blood, being (as it were) picked out and at once precipitated in an insoluble form. I do not see how else the way in which the dots correspond with the vascular papillæ of the gum can be accounted for. I have had one opportunity of examining microscopically the gum of a person who had died while affected by lead-poisoning; and I found that the colour was due to the presence of a multitude of minute black granules. Some of these were aggregated together in the interior of small blood-vessels, the ramifications of which were mapped out by their presence; others were arranged in double lines which I thought probably corresponded with the exterior of other vessels. Thus it seems that the "blue line" is really due to an *excretion* of lead from the blood. And this accounts for the fact (which I believe I was the first to notice) that when iodide of potassium is given to a patient suffering from the poisonous action of this metal, but in whom the line happens to be ill developed or absent, the appearance in question often becomes well marked within a few days, just as lead can then be found in the urine, although none was being excreted previously. I have myself seen at least three instances in which a blue line has thus been brought out by iodide of potassium while the patient was an in-patient in the hospital for symptoms due to lead-poisoning. Dr Frank Smith, of Sheffield, made the same observation independently.

These views appear to afford an explanation of a circumstance, noticed by Mr Tomes, which has given rise to some doubt in regard to the clinical value of the "blue line" as an indication of the presence of lead. I refer to the fact that such a line has sometimes been observed in persons who have not been known to be exposed to the influence of the poison, and who show none of its symptoms. Evidently, if the black granules consist of precipitated sulphuret of lead, the introduction of the smallest quantities of the metal into the blood from time to time might ultimately lead to the formation of a blue line. And there is no one to whose body such minute proportions of lead might not have access. It was at one time supposed that other metals, such as copper or bismuth, might be capable of producing similar appearances. But I believe there is no evidence that this may be the case; and, as regards copper, Dr Clapton has shown that what it really causes is a bluish-green line on the teeth themselves. When the salts of lead are given medicinally in considerable doses, the line often makes its appearance very quickly. Dr Burton met with instances in which it was developed within two days,—one within twenty-four hours,—the quantity of acetate of lead taken by each patient having then been only from fifteen to twenty-four grains. When the blood is richly impregnated with lead, and when the teeth are allowed to be dirty so that plenty of sulphuretted hydrogen is provided, the line may go far beyond what I have hitherto described. The spaces between the dots may be filled up by a uniform black discolouration, which spreads over the gum for some distance from the teeth. The insides of the lips may also present a similar staining; in one patient under my care this was half an inch broad.

It must be added that there are marked individual differences in susceptibility to the influence of lead. Sir Thomas Watson speaks of persons in whom the colic was caused by their sleeping for a night or two in a freshly-painted room; and he contrasts with such cases that of a painter whose first attack occurred when he had followed his occupation for nineteen years.

So far as I know, no explanation can be given of the fact that the introduction of lead into the body should cause colic. The metal is deposited in the tissues; but according to the analyses of Dr George Wilson, of Edinburgh, there is less of it in the intestines than in several of the other organs.*

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* [In a case of marked plumbism which proved fatal under my care with cerebral symptoms, Dr Stevenson discovered in the liver weighing 45 oz., 3·5 grains of lead; in the spleen, weighing 4 oz., ·465 grains; in a deeply pigmented part of the colon, weighing 6 oz., ·246 grains; in the heart, weighing 10 oz., ·054, and in 8 oz. of cerebral matter none at all.—Ed.]

Morbid anatomy.—At the present day I do not know that lead-colic is ever directly fatal, at any rate in England; but formerly patients seem not unfrequently to have died of it. In such cases, and in those in which during an attack death occurs from some other cause, the alimentary canal is generally said to present no morbid change of importance. Several post-mortem examinations were made by Andral, Mérat,* and others. The former found the intestines free from inflammation and neither dilated nor contracted; the latter, however, did find the large bowel contracted, and he also observed the same thing in rabbits which had died of lead-colic. It is true that contraction of the large intestine is not very uncommon in persons who have died from various causes, but if constantly found in those who had had lead-colic it would be significant. Some time ago I made the post-mortem examination upon a patient of Dr Moxon's who had died of heart disease, but who was a painter and had a well-marked blue line. Several years before he had had colic and shortly before his death he complained greatly of pain about the splenic flexure of the colon. At this part of the bowel, and also in the transverse colon, there were scattered several slate-coloured patches which, although not indurated, looked puckered, and which if they were not actual cicatrices, must have resulted from extravasations of blood at some former period. Dr Bristowe showed, at one of the meetings of the Pathological Society, the intestine from a man, also a painter, who had died in St Thomas's Hospital of what was supposed to be colic, and in whom the intestines were enormously dilated and some parts of them black from hæmorrhage into the mucous and submucous tissues. Whether the appearances in these cases had anything to do with the fact that the patient had been exposed to lead-poisoning it would at present be impossible to say.

The *treatment* of lead-colic should be the same as for any other form of the complaint. Sometimes there is considerable difficulty in bringing about an action of the bowels, two or three successive doses of castor-oil with laudanum may be required, and it may even be necessary to add one or two drops of croton-oil. When once a free evacuation has occurred, all the spmptoms generally quickly disappear. The patient should, however, be put through a course of iodide of potassium. As I have already mentioned, this salt possesses the power of eliminating from the body the lead which had been deposited in the tissues, forming with it a soluble compound which is absorbed again into the blood and then excreted by the kidneys. This was long ago established by the observations of Dr Nicholson and Dr Parkes; and I have myself met with one case at Guy's Hospital in which it was demonstrated: the urine had contained no lead before the patient began to take the iodide, whereas the presence of the metal was afterwards detected without difficulty. Probably it is because the lead is apt to remain in the body all through an attack of colic and afterwards that, as is stated by Sir Thomas Watson, the complaint appears to have a special tendency to relapse.†

CONSTIPATION.—Another effect of functional disorder of the intestines is constipation. I have already mentioned this as a symptom of brain-disease and of dyspepsia; and I shall hereafter have to speak of it as occurring in cases of jaundice. But in all such cases it is associated with other symptoms; the constipation with which I have now to deal is a substantive complaint; the patient may show no further indication of being unwell, and if he should do so, his other symptoms are mere results of the defective action of his bowels.

Now, there are great differences in the frequency with which fæcal evacuations are passed by healthy persons. In some people an action of the bowels occurs only at intervals of three or four days, and yet they suffer not the slightest inconvenience. This is not a condition which calls for, or would justify, any medical interference. But in children and young persons, the fæces are often retained for several days at a time, merely because they are careless, or unwilling to face the outside air for a few seconds, or too modest to go to a water-closet the approaches to which are perhaps not altogether private. Great evil may result from this, and all the more because it is unforeseen.

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* 'Traité de la Colique métallique, vulgairement colique de Poitou,' 1810.

† [For other symptoms of plumbism, see vol. i, pp. 447 and 578.—ED.]

Apart from such cases as those last mentioned, in which at first the will is alone concerned, the exciting cause of constipation is either that the peristaltic action of the bowel is too slow or deficient in force, or that the fæces are dried up and hardened to too great an extent, so that they do not readily pass down the intestinal canal. Some writers speak of a "sluggish state of the nervous system" as being the prime cause of constipation; arguing from the undoubted fact that a tendency to the complaint is often inherited and shows itself in many members of the same family. Among the more immediate causes may be mentioned excessive abstemiousness in eating and habitual restriction to a diet which is too exclusively animal. Sedentary habits may also play a part in preventing the bowels from acting properly, particularly in those whose work is mental. Again, affections of the abdominal viscera of various kinds may interfere mechanically with the passage of the intestinal contents, but mere constipation is then merged in a more serious condition—that of obstruction of the bowels—which will be described separately. Only a caution may be given that when a woman suffers from habitual torpidity of the intestines, the possibility must always be borne in mind that this may be due to the presence of a uterine or ovarian tumour, or of a prolapsed or displaced womb.

The evil effects of constipation are due in part to the retention within the large intestine of what should have been voided from the body. Following O'Beirne, I believe that in health the rectum is generally empty, and that when a fæcal mass, even of small size, has once entered this part of the bowel it ought at once to excite sensations which should bring about its expulsion. Among the discomforts caused by slight constipation, not the least is that which results from the passage into the rectum of isolated round pellets which had been moulded in the sacculi of the colon; these may excite a great desire to go to stool, and yet they are passed only after violent straining efforts. Patients in whom this occurs often speak of themselves as suffering from diarrhœa, and only a strict cross-examination may elicit the real state of the case. I remember hearing that Dr Bright was once summoned into the country, in consultation with an eminent surgeon and a general practitioner, to see a lady who had been in vain treated with astringents, being supposed to suffer from a relaxed state of the bowels. He asked to see the evacuations, whereupon a single little hard pellet of fæcal matter was shown to him, and it was at once clear that a purgative was what alone would give relief.

Even if the colon and sigmoid flexure should be the part in which the fæcal masses accumulate, their presence often gives rise to a sense of weight and discomfort and to colicky pains. But in persons who are habitually constipated, the rectum loses its natural sensitiveness; and this part of the bowel may then become impacted with dry hard lumps of excrement of enormous size. Under such circumstances the bowel sometimes becomes irritated and pours out mucus, and this, or fluid fæcal matter, may pass down by the side of the retained masses, so that a condition of diarrhœa may be closely simulated.

Habitual constipation has a marked influence on the general health and spirits of the patient. It is well known to make the tongue furred and the breath foul. It causes a nasty taste in the mouth. It gives rise to feelings of languor and melancholy, and makes the step inelastic and the countenance depressed and haggard.

One cannot wonder, therefore, that those who suffer from constipation are ready to take purgatives almost every day and in constantly increasing doses. One hears of persons who for years have never had an action of the bowels except as the result of aperient drugs, and some of the quack medicines of the greatest repute are employed in this way. Each time that they are taken they cause free evacuations, but they exhaust the susceptibility of the intestine and render it less capable than ever of responding to natural stimuli.

The proper treatment of the complaint is the very reverse of this. One may be obliged at first to prescribe a purge, or to clear out the lower part of the intestine by enemata. But from that time, all ordinary aperient doses should be scrupulously avoided. The best method, I believe, is that which was suggested by Dr Spender in the 'Medical Times and Gazette' for 1870. It consists in the regular administration of a pill containing from one to three grains of sulphate of iron, and about a grain of the watery extract of aloes or of the compound extract of colocynth (or of the compound rhubarb pill). A quarter of a grain of extract of nux vomica or of extract of belladonna may be added; but to the former Dr Spender ascribes no value at all, and he thinks that even the latter is of comparatively little service. At first, the patient should take three pills a day, one after each meal. He should be told that for two or even three days he is not to expect an evacuation, but that when the bowels have once acted they will afterwards be moved more frequently. And now comes the point of importance,—that whenever there is an evacuation of loose consistence, he should instantly decrease the number of pills which he takes. Nothing approaching to a purgative effect should ever be permitted. Very soon two pills a day are sufficient; and a fortnight later, a single one perhaps produces the desired effect. Within another month he is able to do with a pill once or twice a week. If the patient should make a difficulty about taking pills, the best substitute for them is a mixture of the decoctum aloes co. and the mistura ferri co.

I have repeatedly put this plan in practice, and can confirm Dr Spender's testimony to its value. The cases which he reports are very striking. Sometimes I have directed patients at the same time to take a spoonful of olive oil once or twice a day, or to eat brown bread, or to take a glass of cold water on first rising in the morning. The diet indeed should always be carefully attended to, and regular bodily exercise must be taken. And another important point is that the patient should always go to the water-closet at a regular hour each day, and allow the necessary time for the bowels to act. Trousseau insists on this, and rightly observes that without any other treatment it may be successful in overcoming habitual constipation. When it fails, he advises enemata of water, at first with the chill off, afterwards quite cold; or suppositories of cacao butter or soap. But in my opinion it is very unadvisable to employ any measures of this kind, which must tend to render the bowel less susceptible to its natural stimulus. The medicine which Trousseau chiefly recommends is the extract of belladonna.

Intestinal casts.—A rare but very interesting affection of the intestines—generally attended with constipation—is one in which membranous casts of its interior are voided from time to time, often for very long periods. In 1857 Mr Hutchinson showed several specimens of this kind at one of the meetings of the Pathological Society ('Path. Trans.,' ix, 188); they were cylinders several inches or even feet long. Their walls were from one eighth to a quarter of an inch in diameter, yellowish brown, transparent and gelatinous. Under the microscope their surface showed a regular arrangement of round or oval pits, which had evidently corresponded with the mouths of the tubular glands of the intestine. Their substance was almost structureless, but had embedded in it large numbers of epithelial cells. When they had been retained in the rectum, the casts were apt to be changed into hard white round masses, about the size of nutmegs.

More recently Dr Goodhart exhibited some specimens to the same Society ('Path. Trans.,' xxiii, 98) which were almost exactly similar, except that they were solid. Many of them shaded off at their ends into a clear colourless jelly. Both in Mr Hutchinson's case and in Dr Goodhart's there was much complaint of abdominal pain. It does not appear that medicinal or other treatment led to any definite good result.

Hæmorrhage.—Another symptom which occurs in several intestinal diseases is passing blood per anum. The blood itself may be converted into a black tar-like substance; this I believe is always a sign that the stomach was the seat of the hæmorrhage; the affection is then termed melæna. I have already described it at p. 132. In other instances blood is discharged from the rectum almost unaltered; I shall have to refer to such cases, when speaking of intussusception, and of dysentery. In enteric fever it is an important event (see vol. i, p. 195).

DIARRHŒA.—Another result of intestinal disorder is *diarrhœa,*—the discharge of the contents of the bowel in too great quantity, in a fluid condition, and with excessive frequency.

Now, as a rule, affections of the large bowel give rise, not to diarrhœa, but to a complaint which is called dysentery, and which will be described separately. At present, therefore, we may confine our attention to the small intestine. And in this part of the alimentary canal at least three different conditions may arise, either of which is capable of producing diarrhœa; they are the following: an increase in the peristaltic movements, an augmentation in the secretions, and a morbid condition of the mucous membrane. But, in acute diarrhœa at any rate, one can scarcely ever distinguish between these several conditions; and no doubt, in many cases, two or even all three of them are in action simultaneously.

Acute form.—The exciting cause of an attack of diarrhœa, however, is often very definite. Sometimes mental influences give rise to it; it attacks the child who is in dread of being punished, or the man who is about to preach or to lecture. Exposure to cold—getting chilled—is said to be frequently its starting-point, but I do not know that this is really the case. It often follows quickly upon the ingestion of some irritating substance, medicinal or dietetic. In young infants it is very apt to be set up by amylaceous food, which at that time of life cannot be digested, the secretions which convert starch into sugar not being formed during the first few months after birth. Some infants have diarrhœa even if they are fed with the best cows' milk, or with anything but human milk.

Impure drinking-water is also a frequent cause of diarrhœa. In Parkes's work on 'Hygiene' references will be found to numerous observations in which this effect has been produced by water containing suspended or dissolved mineral substances of various kinds, or suspended vegetable matters. Such waters, however, would scarcely be used for drinking purposes except under special circumstances. As a rule, the danger arises rather from the presence of animal matters, especially of excremental origin. Thus diarrhœa has often been traced (as in Croydon in 1854 by Dr Carpenter) to sewage contained in suspension in the water. Probably dissolved solid substances of animal origin may have the same effect, but according to Parkes this has not yet been proved. Dissolved sewer gases, however, are certainly known to be capable of causing diarrhœa. A striking instance occurred in Salford Gaol in 1859. Within four days 266 out of 466 prisoners there were attacked with the complaint, whereas none of the officers nor any member of their families suffered. The water which the prisoners drank was at once examined and found to have a yellowish colour and an insipid taste. The cause of this was that the overflow pipe from the cistern led directly into a sewer and conveyed a most foul stench to the cistern, which was covered in closely with boards. The water supplied to the officers, on the other hand, was clear and refreshing. Both waters came from the same source, being merely stored in separate cisterns.

It is well known that in late summer and early autumn diarrhœa is very apt to prevail epidemically, particularly in large manufacturing towns. Now, it so happened that Dr Greenhow was in 1859 investigating the causation of this form of the complaint. The general conclusion at which he arrived was that in those places in which it prevailed most severely one or other of the local causes could always be traced; either the air was tainted with the products of organic decomposition, especially of human excrement, or the water which the people habitually drank was impure. And the outbreak at Salford, which occurred in the end of September, was naturally regarded as a crucial instance, proving that such conditions are really the cause of epidemic diarrhœa.

Subsequent inquiries, however, have rather tended to throw doubt upon the validity of this explanation of epidemic diarrhœa in general. Dr Greenhow himself observes that as a fatal disease it is almost wholly confined to children under five years. This might, indeed, be attributed to the greater susceptibility of very young organisms, particularly as there is as yet no evidence to show what proportion of attacks of *non-fatal* diarrhœa occur in older subjects. But then it further appears that most of the cases of fatal diarrhœa are in children under one year of age. And Dr Crane, of Leicester, investigated the conditions under which 283 children had been placed who died from this cause in the summer of 1873, with the result that a large majority lived in houses not in bad sanitary condition, and that 107 were wholly suckled, 98 partially suckled, and 78 fed by the bottle alone. Impurity of the drinking-water clearly was not the direct cause of the disease in the 107.

Dr Buchanan has lately made some remarks upon this subject; and, while not denying that summer diarrhœa has associations with filth, he is evidently inclined to think that it is really due to a specific materies developed by the influence of heat at a particular season. One point on which he insists is that whatever may be the heat of the weather before July it does not cause epidemic diarrhœa. In May or June the temperature may average 60° or 62°, or be even higher, and yet no increase of diarrhœa will result. Yet the connection of the disease with autumnal heat is certain, for the mortality from this cause is much greater in hot than in cool summers. A curious circumstance, to which Dr Buchanan draws attention, is that the disease seems to be of modern introduction, so far as can be learnt from the old bills of mortality. At the beginning of the present century there is no direct mention of it, under whatever name, nor can any special mortality among infants be traced as having occurred in the summer or autumn months. But for many years past epidemic diarrhœa has been a very fatal disease, sweeping away thousands of lives annually.

The diarrhœa set up by the causes hitherto mentioned is an acute disease; one that may run its course in a few hours, and that perhaps never lasts more than a week or ten days—if we except those cases in which such a complaint may be kept up by the repeated ingestion of irritating food or bad water. Another form is that which occurs as a complication of acute diseases, particularly of puerperal fever.

When acute diarrhœa proves fatal the *anatomical changes* discovered after death are generally very slight. The inner surface of the small intestine may be reddened and lined with mucus, or softened. But as in the case of other mucous membranes vascular injection probably often disappears after death, so that it may not be seen at an autopsy, even though it existed during life; and, on the other hand, there are great doubts whether softening, on which French pathologists formerly laid great stress, is not a cadaveric change.

Sometimes, however, the mucous membrane of the small intestine is attacked with a much more marked and severe form of inflammation. A *diphtheritic* state, especially marked in the precincts of the valvulæ conniventes, may be found throughout a large part of the ileum or jejunum. In all such cases relaxation of the bowels is a principal symptom, and I do not know that at the bedside they can be distinguished from the more severe instances of ordinary diarrhœa, although the pathologist, finding diseased conditions sufficient to account for death, is naturally disposed to place them in a separate category.

Chronic diarrhœa.—Another and a very different kind of diarrhœa runs a chronic course, lasting for months or even for years. The intestine then often presents appearances which are regarded as indications of chronic catarrhal inflammation. Thus, slaty or black patches may be seen in its mucous surface, or minute black dots, corresponding with the solitary follicles. Its coats may be thickened and it may be lined with a viscid opaque mucus. These changes are particularly well marked when they are secondary to mechanical congestion, as in cases of heart disease, or of liver disease, both of which are commonly complicated with diarrhœa.

Sometimes, however, one may be unable to detect any definite change in the intestine although the diarrhœa had been present for a great length of time before death. I once made a post-mortem examination in the case of a gentleman who had come home from China with what is termed "white flux," in which there is constant diarrhœa with discharge of matters devoid of bile. At the time of his death the complaint had lasted some years, and the only morbid appearance which I could discover was an extreme thinning of the coats of the intestine. In infants, in whom chronic diarrhœa is a frequent cause of death, one seldom finds any pathological change. Dr Eustace Smith mentions that ulcers are sometimes present in the large intestine; but, as he says, these are probably secondary and the result of the irritation set up by acrid matters which had been secreted by the bowel higher up.

Tubercular.—But there are other forms of disease in which diarrhœa is a principal symptom, and in which changes in the bowel leading to ulceration, constantly occur. Enteric fever might be cited as a case in point, but both its symptoms and the intestinal lesions which accompany it have been fully described already. The affection on which I must now lay most stress is of a *tuberculous* nature. The earliest stage in which this can be recognised is that in which opaque yellowish spots are seen in or beneath the mucous membrane. They doubtless are the result of caseation of the tissue of solitary follicles, or of the follicles of a Peyer's patch the lymphoid tissue of which had before undergone augmentation. And I think that in all probability the augmentation arises by a formative process. One could, indeed, conceive that it might be due to one of simple inflammation. But after careful investigation of the question I have come to the conclusion that in the post-mortem room one seldom or never finds caseation of solitary follicles in the intestine without tuberculous lesions being present in other parts. The next step in the development of the affection is that the mucous membrane covering the little yellow spots breaks through, and a small circular ulcer is formed. This almost at once acquires a smooth rounded edge, which is indurated, so that to the finger it feels almost like a rim of leather. The increase in size of the ulcer always takes place chiefly in a direction transverse to the axis of the bowel. Thus its form becomes elliptical, or even roughly quadrangular, and it may become so large as completely to encircle the bowel. Its floor is generally formed by the muscular coat, which is thickened by inflammatory products and may still have some yellow cheesy granules adherent to its surface. The subserous tissue and serous membrane also become thickened and opaque, and these changes, and the presence of an injected zone of blood-vessels round the ulcer enable its seat to be clearly recognised on the outer surface of the intestine. A more important character still is the presence, in many cases, of distinct tuberculous granulations in clusters, or forming long ridges, which are believed to correspond with the sheaths of lymphatic vessels, or (according to Rindfleisch) with the smaller arteries. Tuberculous ulcers are more common in the lower part of the ileum than in any other part of the intestine; they are often very numerous, and just above the ileo-cæcal valve they may form extensive patches of very irregular shapes. Sometimes, however, only one or two of them are present, and they may be confined to the upper part of the ileum, or even to the jejunum; or those which occur there have much more marked characters than any which can be found lower down; or, again, they may be seen only in the cæcum or the colon, which portions of the bowel are indeed very liable to be affected in common with the ileum. Tuberculous ulcers of the intestine are probably never seen in the post-mortem room without the lungs being likewise affected with active tuberculous disease. And their clinical importance is generally altogether subordinate to that of the pulmonary phthisis, to which the patient in reality succumbs. They do, indeed, afford an explanation of diarrhœa, when it is present, but in many cases this and all other symptoms of intestinal lesions are wanting, so that the autopsy alone reveals the fact that such ulcers have been forming. Sometimes, however, before any symptom or auscultating sign of phthisis is discoverable, a patient suffers for a very long period from a diarrhœa, and this is ultimately proved to have been due to a tuberculous affection of the intestine. Trousseau quotes Chomel as having especially insisted on the importance of fever and night-sweats as indications of the presence of such an affection.

It is said that tuberculous ulcers sometimes heal and that their cicatrices may produce stricture of the bowel; this I shall discuss when I am speaking of intestinal obstruction. And, under acute peritonitis, I must mention cases in which ulcers of this kind have given way into the serous cavity. Much more commonly the affected coil of intestine becomes adherent to a neighbouring coil, and an opening forms between them; in this way a series of communications between one part of the bowel and another may be formed.

Lardaceous.—Another cause of chronic diarrhœa is the presence of lardaceous change in the intestinal mucous membrane. This, I believe, never occurs without other organs being affected, and in a marked degree. It may be caused either by syphilis or by protracted suppuration, of which one instance is that which accompanies chronic pulmonary phthisis. It may be worthy of note, so far as concerns syphilis, that Trousseau lays stress on it as an occasional cause of chronic diarrhœa, but in the particular case to which he refers the affection can hardly have been lardaceous, for the symptoms yielded to mercurial treatment. The peculiar change is said by Dr Moxon to begin in the walls of the minute arteries, and to spread from them into the tissues around. To the naked eye the mucous membrane presents an appearance which one can more easily recognise than describe; Dr Moxon compares it to wet washleather. Iodine stains it of the colour of dark walnut wood. Peyer's patches are generally less affected than the rest of the mucous membrane.

Malignant.—Yet another cause of chronic diarrhœa—comparatively a very rare one—is the development of a new growth in the intestinal walls. A carcinomatous ulcer sometimes gives rise to this symptom, but very seldom, for such ulcers, almost without exception, narrow the bowel and cause obstruction instead.

More frequently diarrhœa is set up by a form of lympho-sarcoma, the distinctive characters of which were, I believe, first pointed out by Dr Moxon. It may invade a large extent of the intestine and completely surround it at various points, but always with the effect of making it wider than natural. It constitutes a white, soft, medullary growth, and has little or no tendency to ulcerate. A marked instance of this affection occurred in a child who died under my care in the Evelina Hospital; the growth everywhere seemed to have entered the coats of the intestine along the line of its attachment to the mesentery.

Symptoms.—Of the symptoms of diarrhœa there is little to be said. The discharges may consist of a fluid fæcal matter just like that which the small intestine normally contains at a certain period after digestion of food. It may be of a bright yellow colour, or more or less brown. In infants, the evacuations of diarrhœa are often green. This was formerly supposed to be a result of the administration of calomel, but it is now known to be due to changes in the bile pigment which are independent of any such cause. We shall hereafter find that bilirubin (as it is called) is apt to turn green when acted on by an alkali; and the statement has been made that green diarrhœal matters are always alkaline, but, as Kühne pointed out, this is a mistake. In adults, the evacuations often look as if they consisted of pure bile, and are said to respond to the tests for the biliary constituents much better than ordinary fæces. In other cases diarrhœal discharges are pale and watery, and they may even approach in character the "rice-water stools" of cholera. Under the microscope crystals of triple phosphate can often be detected in the matters voided from the bowels in all forms of diarrhœa. Mucus is sometimes present in considerable quantity, but pus can seldom be identified, either with the naked eye or microscopically. Hæmorrhage forms no part of mere diarrhœa. I do not know that blood can ever be detected, even when there is extensive tuberculous ulceration.

Acute diarrhœa is generally accompanied by some colicky pain, by sickness, and by slight tumefaction of the abdomen. In chronic diarrhœa these symptoms are commonly absent; the abdominal walls often become deeply sunken and retracted. In infants prolapse of the rectum is very apt to occur as a complication, and the anus generally becomes sore and excoriated.

Mild cases, in adults, are attended with little or no disturbance of the general health. But in young infants even very slight diarrhœa may give rise to great depression of the vital powers, indicated by coldness of the surface, by dark pigmentation and sinking in of the spaces round the eyes, and by depression of the fontanelles. This last is a most valuable sign, and must always be borne in remembrance; it often gives a warning of danger at a comparatively early period, when the child would otherwise seem to have but little the matter with it, and when neither the pulse nor the respiration is accelerated. I have already mentioned that in infants the disease is very apt to terminate fatally. Death may also occur in old people and even in adults, if exhausted by previous disease or privation; the symptoms are then always those of collapse. Such cases, in which the evacuations are generally profuse and watery, commonly receive the designation of "choleraic diarrhœa" or of "English cholera," and these names regularly appear in the returns of the Registrar-General every autumn. Whether English cholera ever proves fatal to a healthy grown-up person is a question which I have discussed in speaking of the Asiatic or epidemic form of the disease (vol. i, p. 306).

The *treatment* of diarrhœa is a matter which requires judgment on the part of the practitioner, and for which detailed rules can hardly be laid down; but some general principles may be stated.

In acute diarrhœa of any severity, the patient must be kept in bed, and only a very bland diet should be allowed. Sometimes it is desirable to give an aperient with the object of clearing away any irritating substances that the intestines may contain; and a dose of castor-oil with a little opium may be administered; or, what is perhaps better, a scruple of Gregory's powder, or of some other preparation of rhubarb. But, as a rule, all that is in the bowels is being swept away by the diarrhœa itself. The best medicine is then a stomachic with a little alkali. A formula which I have used, and seen used largely, consists of a scruple of carbonate of soda, twenty minims of aromatic spirits of ammonia, and an ounce of peppermint water; this may be repeated every two or three hours. Another valuable remedy, much employed by continental physicians, is the subnitrate of bismuth.

It is not, I think, advisable to prescribe opium, or even morphia, in acute diarrhœa, at least until other remedies have had a fair trial. Nor should astringents be given at the commencement of the attack. On the other hand, in chronic diarrhœa, astringents are often very valuable, and may suffice of themselves to cure the patient. Hæmatoxylum, krameria, kino, catechu; the extract of Indian bäel; the compound chalk powder and the aromatic confection; alum, pernitrate of iron, nitrate of silver, and sulphate of copper; each of these may be used. Many medical men employ sulphuric acid largely, particularly in children; others believe it to be useless.

Sometimes, however, each astringent seems to lose its effect after the patient has been taking it for a few days, and one is then obliged to prescribe them all in turn. This is particularly apt to occur when there is tubercular ulceration or lardaceous disease of the intestines; but diarrhœa may last for a very long time without there being any evidence of organic change in the bowel. In some cases of this kind opium is very serviceable; and it may be continued in free doses for several months without appearing to affect the patient injuriously in any way. For cases of chronic intestinal catarrh, Trousseau speaks highly of the arseniate of soda, and I am quite prepared to believe that this may be useful from what I have seen of the influence of arsenic in other catarrhal affections.

A method of treating diarrhœa, which in infants often succeeds, consists in giving no food whatever except raw meat, finely grated into a pulp and mixed with powdered sugar or currant jelly to make it palatable. Trousseau calls this "conserve de Damas" for the sake of mystification. He relates the case of a young lady who had had intractable diarrhœa for six months, and who was quickly cured by raw meat. To remove any prejudice he gave directions that each slice should be exposed to a strong fire for a few minutes, but that the part of it which had been acted on by the heat should be entirely cut away before it was given to the patient.

Enteritis.—I have hitherto avoided the use of the term enteritis, and it appears to me that there is no distinct affection which requires to be described under that name. Cases do, indeed, occur in which an intense inflammation extends from the mucous membrane of the intestine to the other coats, but at the bedside such cases fall either under the head of severe diarrhœa or under that of dysentery. On the other hand, it is not uncommon for a patient to present symptoms which clearly indicate that besides peritonitis he has inflammation of some part of the bowel, but after death, even if not during life, the affection seems always upon careful examination to be referable to some form of mechanical obstruction, or at least to what will presently be described as volvulus. Thus to give an account of *enteritis* as a substantive disease is to deal over again with cases which have to be fully considered under other heads. I believe one may say that there is no affection which, both clinically and in the post-mortem room, deserves that name, and which is in fact so designated both by the practical physician and by the pathologist.

Perityphlitis or Typhlitis.—One particular part of the intestine, however, is very liable to be attacked by inflammation, and the disease which results is certainly one requiring a special description. That part is the cæcum, and the disease is commonly known as perityphlitis or typhlitis. Systematic writers, indeed, mention these as two distinct affections. The former, they say, is an inflammation of the connective tissue behind the cæcum. It runs a chronic course and comparatively seldom destroys life, except as the result of protracted suppuration. The latter includes the rapidly fatal cases of perforation of the appendix vermiformis.

Dr Wilks, however, has repeatedly expressed to me the opinion that both in "typhlitis" and in "perityphlitis" the disease begins in the appendix, and that variations in the intensity of the morbid process there are the real cause of the supposed distinction between them. And, so far as I can learn, all the evidence which morbid anatomy affords points strongly in this direction. Dr Theodore Williams has recorded in the 'Pathological Transactions' a case in point. A man who was being treated for pleurisy was attacked with pain in the right iliac fossa, vomiting, constipation, &c.; a tumour of the size of an orange could be felt in the right iliac fossa. Some days elapsed, and all the symptoms were subsiding, when pneumothorax supervened and he died. He had passed several dark and offensive motions containing scybala, and in consequence the swelling had diminished in size until it could scarcely be detected. Thus the case was a perfect example of what would ordinarily be called perityphlitis and be regarded as the result of the accumulation of fæcal matters in the cæcum. For it appears to me that in the common use of the term "perityphlitis" there is in reality no intention to limit it to cases in which the connective tissue behind the bowel is the exact seat of the disease, but rather an unacknowledged feeling that the term has a wider signification than typhlitis, and means merely that the disease is "about" or "in the neighbourhood of" the cæcum. However this may be, the real nature of Dr Williams's case would not have been cleared up if the patient had not died accidentally from another cause. And then it was found that the cæcum was surrounded by adhesions, but that there was a small collection of purulent matter round the appendix vermiformis in which Dr Powell found a minute perforation. Outside the aperture lay a small mass of hardened fæcal matter. Dr Wilks has mentioned to me two cases which have come under his observation, and which tend to establish the same conclusion. In each of them the patient had a comparatively mild attack and recovered; afterwards he was seized a second time with the disease and died, and a post-mortem examination then showed that the disease had originated in the cæcal appendix, and that this was perforated.

The name typhlitis therefore seems preferable to perityphlitis. The affection spreads from the mucous to the serous coat of the appendix, either by a process of ulceration or by one of sloughing; in the latter case the end of the tube, or one side of it, may be found in a gangrenous state. The cause of the inflammation is generally the presence of a concretion. This may be of the size of a pea, or as large as a plumstone. It sometimes consists of a substance like wax, but much more frequently it is composed merely of hard dry fæcal matter, mixed with mucus and containing a large proportion of earthy salts. A mass of this kind may look very like the stone of a cherry or some other fruit, and has often been mistaken for such a substance. Indeed, supposed "foreign bodies" from the appendix have so frequently been found on examination to be of fæcal origin that many pathologists are disposed to doubt whether typhlitis is ever set up by such a thing as a fruitstone. It is, however, certain that seeds, pills, bristles, pieces of bone, shot, &c., have been found in the appendix, and that some at least of them have led to its perforation.

But sometimes ulceration of this part of the bowel, penetrating its serous coat, has been tuberculous; and in yet other cases no exciting cause for the inflammation can be discovered. It is possible that the walls of the appendix may have given way as the result of its distension with fluid, for such a condition is now and then met with, the opening into the cæcum being closed. Dr Wilks saw a case in which the appendix was dilated to the size of the ileum, and distended with three or four ounces of white odourless mucus.

The extent to which the inflammatory process spreads over the peritoneal surface varies greatly in different cases of typhlitis. Sometimes it lights up almost instantaneously throughout the whole serous cavity. There is then during the early part of the case no possibility of determining the fact that ulceration or sloughing of the appendix formed the starting-point of the disease; one can only set it down as an instance of acute peritonitis, the cause of which is unknown. Of such cases I shall speak again further on. When they terminate favourably it becomes possible after a time to detect a hard swelling in the right iliac fossa, and this clears up their nature.

In other instances the symptoms of acute peritonitis are wanting or but little marked. Their main clinical feature depends upon the inability of the intestinal contents to pass through the affected part of the bowel. They may, I believe, be undistinguishable at the bedside from cases of actual mechanical obstruction, and I shall have to refer to them again when describing that condition.

Thus the cases of typhlitis that can be satisfactorily diagnosed form only a moiety of the whole, and, as a rule, they are the milder cases,—those in which the inflammation does not spread beyond the serous covering of the cæcum and the parts immediately adjacent. Among the symptoms is pain, referred mainly to the right iliac fossa. This is generally in part paroxysmal; it is often of extreme severity; it is associated with more or less tenderness, the latter being sometimes so marked that the patient cannot bear even the slightest touch. Nausea and vomiting are generally present, and also constipation. The amount of constitutional disturbance is very variable. But the principal symptom is the presence of an ill-defined rounded swelling extending upwards from the iliac region towards the right loin. This is doubtless formed in part by the thickened coats of the affected portion of bowel, but in much greater part it is due to the accumulation of fæcal matters within it. The size and form of this swelling may vary from day to day. If the disease subsides it gradually disappears.

Typhlitis is very much more common in males than in females. Out of ten consecutive cases at Guy's Hospital, eight were in boys or men. It is a disease of an early period of life; in seven of the ten cases the patients were between thirteen and twenty-one years old.

The course of typhlitis depends, I believe, very largely upon the *treatment* which is adopted. I have, indeed, a strong conviction that no case in which the disease can be diagnosed as being seated in the cæcum ever terminates fatally if judiciously managed. The essential points are that the patient should be kept perfectly at rest, that his diet should be strictly limited to slops, that he should not be allowed to take a single dose of aperient medicine, and that opium should be given freely. The treatment should in fact be just such as will hereafter be recommended for acute peritonitis in general. And when the attack subsides, the greatest care must be taken to prevent a relapse. Even then the action of the bowels should be solicited by enemata only, and never by medicines taken into the stomach; and the restriction to fluid food should be continued for several days longer than seems at first sight to be necessary. I have repeatedly seen relapses occur from disregard of precautions that I had believed myself to have insisted on with sufficient emphasis to ensure their being attended to. The disease, indeed, is one which is very apt to recur, even at considerable intervals of time. I know of more than one instance in which repeated attacks have taken place, with a few weeks or months between them, until at length there has been one so severe as to place life in imminent danger. Probably it was because the patient then submitted to being kept in bed for a considerable time, and observed the greatest possible care during convalescence, that in each case this alarming attack was the last which occurred.

The occasional termination of typhlitis in abscess will be described with other forms of local peritonitis.

Even when typhlitis presents itself clinically under the guise of intestinal obstruction or of diffused acute peritonitis I believe that it scarcely ever destroys life if judiciously treated. There may be cases in which a considerable part of the appendix sloughs away, and in which death is inevitable. But I only know of one fatal case as having occurred within the last five years at Guy's Hospital, if I may exclude cases in which death took place a few hours after admission and in which purgatives had been given freely.

DYSENTERY.—I have next to speak of a disease the symptoms of which are very different from any that I have yet had to mention. The patient has frequent desire to go to stool, but passes little except mucus or blood; at the same time he has a severe burning sensation at the anus; he further complains of griping pains in the abdomen, in the course of the large intestine.

A passage in Herodotus clearly alludes to this malady;* and the term "dysentery" is used by Hippocrates, who correctly refers it to ulceration of the intestine. We shall, indeed, hereafter see that the symptoms I have enumerated are not by themselves sufficient to characterise it, for they may be caused by different affections of the large bowel. Hence it will be convenient that I should first describe the morbid appearances which belong to the disease.

Anatomy.—The morbid change which characterises dysentery consists in an inflammatory process which has its principal seat in the mucous membrane, and which is not limited to any one part, but spreads more or less widely throughout the whole length of the large intestine, and even into the lower part of the ileum. It is said by Aitken and Maclean that the small intestine is attacked only in that variety of the disease which they term "scorbutic," but I do not know on what evidence this statement rests.

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* [Of the army of Xerxes during its retreat from the invasion of Greece: *ἐπιλαβὼν δὲ λοιμός τε τὸν στρατὸν καὶ δυσεντερίη κατ'ὁδὸν διέφθειρε.* lib. viii, cap. 115.—ED.]

Certainly cases have occurred in Guy's Hospital in which the last few feet of the ileum have been diseased, in conjunction with the whole length of the large intestine, but in which no indications of scurvy have been present. So that I believe extension to the small intestine may occur in any severe case of dysentery.

In some instances all the large bowel, from the rectum to the cæcum, shows morbid changes of the same kind and in the same stage. But in others the disease is more advanced, or more severe, in one part than in another. Commonly the rectum is the seat of the most intense changes, and these gradually diminish towards the cæcum; but sometimes the reverse is the case: this was noted by Sydenham. In other cases the flexures are said to suffer more than the intervening parts of the bowel.

The appearances presented by the affected parts in dysentery are exceedingly varied, but most writers are now agreed that the processes concerned in their production may be reduced to two. Virchow designates these respectively "catarrhal" and "diphtheritic;" and very good descriptions of them have recently been given by Heubner, of Leipzig. In the *catarrhal* form, the mucous membrane at first shows lines and patches of a dark red colour, with points which are almost black. The summits of any ridges or folds projecting into the interior of the bowel are more injected than other parts of the surface. The mucous membrane is lined with a rather thick layer of mucus streaked with blood. It is itself swollen, and so is the submucous tissue. In the earliest stage of the disease, all that the microscope reveals is a dilatation of the minute blood-vessels, which are gorged with blood. Soon, however, inflammatory products are poured out. The mucous membrane is now still more swollen; it becomes less uniformly reddened; the solitary glands are enlarged and appear as white points with red rings round them. The submucous tissue is increased to from three to five times its normal thickness; even the muscular coat is swollen and thrown into folds. Under the microscope all the tissues are seen to be infiltrated with pus-cells, which are also present in large numbers in the mucus lining the interior of the intestine. In the submucous tissue the pus-cells occur chiefly in the spaces round the blood-vessels. The solitary follicles are markedly increased in size; the lymph sinus which surrounds each of them is wide, but does not itself contain pus-cells; such cells, however, are collected in large numbers under the thin mucous membrane which covers the glands, and which evidently was preparing to give way and rupture.

In former days there have been vehement discussions as to the importance of these changes in the solitary follicles, in relation to the changes in the rest of the mucous membrane. In 1843—1844 Dr Parkes examined the intestine very carefully in numerous fatal cases of dysentery, and stated that the earliest lesion was the alteration in the follicles; and Dr Baly arrived at the same conclusion from his investigations at Millbank Prison. Even before this the question had been mooted; Cruveilhier had insisted that the disease was not a follicular inflammation, and had gone so far as to say that the solitary glands had no share in it. Since the publication of Dr Parkes's observations, the objectors to his views have rather taken up the ground that the white granules visible in the early stage of dysentery are not really solitary follicles, but new formations. The right opinion, however, appears undoubtedly to be that which has been stated in the previous paragraph; namely, that the solitary glands are enlarged in the early stage of the catarrhal form of dysentery, but that this is, after all, only a part of a general process of inflammation which affects the whole mucous membrane, and even the deeper parts of the intestine.

A little later, the mucosa softens down beneath the increasing infiltration of pus-cells, and ulcers are formed. A peculiar appearance is now produced by the changes round the solitary follicles. Their roofs give way, and minute round holes are produced, each of which leads into a small cavity having in its interior the substance of the follicle, which is isolated from its attachments and in fact forms a small slough. The destruction of the mucous membrane, however, is by no means confined to those parts of it which correspond with the glands. It rather takes place between them, so that for a time each orifice is surrounded by a little ring, which appears to be raised, and looks like a deposit upon the surface instead of being a remnant of the original tissue. Even when the ulcers have increased in size, and run together so as to form large patches, there remain irregular islands of still undestroyed mucous membrane, which are of a bluish-red colour and covered with grey or greenish layers of tough mucus. The ulcerated surfaces have themselves a yellow or yellowish-red colour; their floor is formed by the submucous tissue.

Widely different are the appearances in the other form of dysentery —which, in obedience to the teaching of Virchow, all modern German writers call *diphtheritic* (cf. vol. i, p. 274). And here I must for a moment digress to point out that this term, "diphtheritic" has now two meanings, which must be carefully distinguished from one another. I have spoken in the first volume of *diphtheria* as a specific disease, generally affecting the throat. But, in Virchow's sense, a "diphtheritic" inflammation of a mucous membrane or of the skin has not necessarily anything to do with that disease; it is a morbid change of a particular kind, probably dependent merely upon intense irritation of the part. This pathological condition requires to be distinguished by a name of its own, and one seems to have no alternative but to employ that which Virchow has suggested for it, although this double meaning of "diphtheritic" is undoubtedly apt to create confusion in the mind of an English reader.

In the *diphtheritic* form of dysentery the whole thickness of the intestine is from the first affected in a marked degree. Even the serous surface is intensely injected, so that it is of a dark bluish-red colour. The bowel feels hard as well as massive. In its interior there is a thin reddish fluid, or, in some parts, a little fæcal matter. Its lining is of a greyish-red colour and here and there exhibits what looks like a raised deposit on its surface. This, in its earliest stage, is present only on the summits of the ridges formed by the mucous membrane. It may be seen forming transverse lines in the ileum, the lower part of which is commonly affected in this form of the disease. Lower down, in the cæcum, the seeming deposit becomes more extensive; and in the colon it occurs in large patches, or may even occupy the whole surface; being, however, broken up into plates by deep grooves or fissures. The parts affected in this way look dry and granular; to the touch they feel rough and hard. Their colour varies to some extent with that of the intestinal contents, which possess the power of staining them. Thus they may be yellowish, greenish, dark red, or even black. On making a section through the intestine one finds that it is enormously thickened; the muscular layer is much thicker than natural and folded in and out. But the most striking change is in the internal coat. Instead of the dry rough substance above described being a deposit on the surface of the mucous membrane, it is now seen to take the place of that structure and perhaps even of the submucous tissue also. The whole thickness of the intestinal wall within the muscular coat may thus be made up of a tough, homogeneous, yellowish-red material, which offers considerable resistance to the knife, and in which the natural strata can no longer be recognised. Even under the microscope one can hardly make out the original elements of the tissues. One sees nothing but a mass of extravasated blood, of hard amorphous fibrinous exudation, and of pus-cells in greater or less number. But Heubner states that in very fine sections, lines of epithelium arranged in double rows may be identified as the remains of Lieberkühn's tubules.

According to some recent observers, however, the fibrinous material, which I have spoken of as amorphous, contains elements of great importance. It is highly granular, and they are of opinion that these granules are really bacteria.

It is at any rate evident that the apparent "deposit" or "membrane" in the diphtheritic form of dysentery is really formed by the exudation of fibrin and the extravasation of blood into the tissues themselves. This process can have but one termination—the death of the affected structures. Accordingly, whenever there has been time for the occurrence of further changes, eschars are found; and at a still later period these break down into shreds or detritus and are cast off, exposing deep and ragged ulcers of dark green or brown colour.

The changes presented by the intestine in *catarrhal* and in *diphtheritic* dysentery are so different in appearance that one would at first sight be disposed to regard them as belonging to different diseases. It is, however, certain that they merely indicate different degrees of severity in the morbid process. For they are very frequently found side by side in the same intestine, the more intense inflammation being present in those parts (such as the rectum or cæcum) which were earliest attacked. And this being so, the fact that the solitary follicles appear not to be specially affected in even the earliest stage of the diphtheritic form affords strong corroborative evidence in favour of the view that these glands are not the seat of any primary or special change, even in the catarrhal form of dysentery.

In severe cases, abscesses also arise in the submucous tissue, and these may burrow, so that undestroyed parts of the mucous membrane over them are detached in the form of bridges. When pressure is made upon these, the pus may exude at several distant spots. The inflammation extends at one or more points through the muscular coat, and perhaps penetrates to the serous membrane, so that perforation of the bowel occurs, with consequent peritonitis; or the connective tissue at the back of the bowel may be reached by the ulcerative process, the result being that a fæcal abscess is formed. I have seen one case in which such an abscess formed a large tumour filling the left side of the abdomen, extending into the psoas muscle, and into the spleen (which was sloughing) and denuding the ilium of its periosteum over a considerable space.

It is probable that the most intense form of dysentery, in which the whole large intestine and the last few feet of the ileum are uniformly affected, is necesarily a fatal disease. But if the morbid changes in the bowel be not too extensive and severe, recovery may take place, even though in some parts they may have assumed a diphtheritic character. In the catarrhal form, the inflammation probably often subsides before any breach of surface has occurred. When ulceration takes place, and the ulcers subsequently heal, a thin membrane is formed over their surface, which is at first depressed below the level of the parts that had been unaffected; but this difference gradually becomes less obvious, and ultimately disappears. In the diphtheritic form of dysentery the ulcers left by the separation of the sloughs become covered with granulations; their undermined edges adhere to the submucous tissue, and thickened and irregular cicatrices gradually develop themselves. The cicatrices which follow dysentery are always of a dark grey or even black colour, which probably results from a chemical reaction between the colouring matter of blood extravasated during the course of the disease, and the sulphuretted hydrogen, which is one of the gases contained in the interior of the bowel.

In many cases, however, there is no definite termination of the morbid process, either in the death of the patient, or in his recovery. The disease passes into a chronic form, and may continue for months or even years. The ulcers may then remain unhealed, or fresh ones may form in succession as others cicatrise over. But, as Professor Maclean points out, it is a mistake to suppose that unhealed ulcers necessarily remain present so long as symptoms of dysentery in the chronic form persist. Cases are often observed in which not a single breach of surface is discoverable on post-mortem examination. Numerous black cicatrices may of course be seen, but the essential pathological change is an atrophy of the coats of the bowel, the glandular structures having disappeared, and the coats being so attenuated as to be quite transparent.

Sporadic and endemic forms.—Probably there is no part of the world in which dysentery does not sometimes occur *sporadically*, but in London it is now decidedly a rare disease. There are, however, countries in which it is exceedingly prevalent, so that it may be said to be *endemic* there; and even in regions where it does not thus prevail it sometimes affects large numbers of persons at the same time, in fact spreading as an *epidemic*. As a general rule, the disease is much milder when sporadic than when endemic or epidemic. Slight cases may indeed occur under all circumstances; but whenever many individuals are attacked simultaneously, dysentery is apt to assume a severe type. Heubner, indeed, is disposed to think that a primarily diphtheritic dysentery never occurs sporadically. But among fourteen or fifteen cases of acute and rapidly fatal dysentery that in the course of the last twenty years have from time to time occurred at Guy's Hospital, there have been several in which the inflammation showed the most marked diphtheritic character. It is therefore evident that, so far as concerns the anatomical changes in the intestine, no absolute distinction exists between sporadic and epidemic dysentery.

Ætiology.—The origin of *sporadic dysentery* is commonly attributed to the ingestion of irritating articles of diet, such as unripe fruit, decomposing meat, or bad water. But Trousseau and others have disputed the correctness of this opinion. It has been urged that when anything which disturbs the intestine is swallowed, active peristaltic movements are excited which expel it from the body, and that ingesta are not likely to leave the small intestine unaffected and to exert an irritant action first upon the cæcum and colon. But it is well known that this very thing occurs in cases of poisoning by bichloride of mercury, in which violent inflammation and even ulceration of the cæcum and colon have repeatedly been observed although the small bowel has escaped entirely. Moreover, there is some reason for thinking that one factor in the causation of dysentery may be a habitual torpidity of the large intestine. This is a point on which Virchow lays stress; he remarks that the cæcum and the flexures of the colon, which are particularly liable to be affected by the disease, are also especially apt to become loaded with fæcal masses. And it is evident that if there is any irritant substance among the intestinal contents its action must be favoured by their retention in the bowel as the result of imperfect peristalsis. Now, Annesley long ago pointed out that in India the disease often commences with the characteristic signs of morbid accumulation in the large bowel. And in connection with this, the suggestion of Dr Dickinson may be remembered that certain transverse ulcers in the colon, which are not uncommonly found in the bodies of those who have died inmates of London hospitals, and which have been known to perforate the bowel or to cause a fæcal abscess, are due to irritation from the fæces. Another cause to which sporadic dysentery has been attributed is cold. Heubner alludes to a case which occurred in a washerwoman who had been standing for a long time with the clothes about her body wet through.

As regards the causation of *endemic and epidemic dysentery* very little is as yet certainly known. The disease may be said to have its home in the tropics. On each side of the equator, to about 35° or 40° of south or north latitude, there are in all parts of the globe territories in which it prevails, but it is a great mistake to suppose that this is necessarily the case in every country which has a hot climate. Hirsch mentions the peninsula of Gujerat in India, and Senegal in Africa, as regions in which the heat is intense, but in which there is no dysentery. So, again, Singapore is said to be free from the disease, which yet exists in all other parts of the peninsula of Malacca. The rainy season is generally the period of the year at which dysentery is most apt to prevail. The alternation of hot days and cold nights has been supposed to have a special influence in producing it.

In temperate climates, epidemic dysentery occurs, at the present day, chiefly in camps and armies. It was very fatal in 1854 among the British troops engaged in the Crimean war, and, in 1870, among the soldiers who fought in the campaign between France and Germany.

Even in time of peace the large cities of Europe were formerly liable to epidemic dysentery, and Paris suffered severely from it as lately as in 1859, after having been free for a hundred years. In London, in the seventeenth century, it is believed to have caused from 1000 to 4000 deaths annually; in the following century it gradually disappeared. In Millbank prison, however, small epidemics of the disease were of frequent occurrence until a few years back. As in the tropics, so in temperate climates, the autumn is the season at which the disease is most apt to break out. The years of its prevalence have sometimes been exceptionally hot, but this has not invariably been the case.

Dysentery occurs in persons of all ages. In this country it is not uncommon in infants who are brought up by hand. When it is epidemic, it attacks especially those who are weak or old, or whose health is impaired by intemperance.

The conditions which are concerned in the production of epidemics of dysentery have been studied with the utmost care and attention; but, as one might indeed have anticipated, it is scarcely ever possible to make out which of them is really the exciting cause of the disease.

Certain facts appeared at one time to point strongly to the conclusion that it was a telluric poison, like that which generates intermittent fever. Thus it has long been known that even in temperate climates, and still more in the tropics, the countries in which dysentery prevails are also those in which ague is common. Indeed, the two diseases frequently occur together in the same patient at the same time; and Dr Aitken remarks that if a boat's crew be sent ashore in a tropical climate, and exposed to paludal miasmata, the probabilities are that of the men returning on board some will be seized with dysentery and others with remittent fever. So again, the gradual extinction of dysentery within the last two centuries in England has coincided with a very great decrease in the amount of ague throughout the country, and with its complete disappearance from certain parts. It was, indeed, known that the very same miasm could not be the cause of both diseases, for dysentery prevails in many places in which there is no intermittent fever. Still it appeared probable, until quite recently, that the real cause of the former disease was some poison allied to that which produces the latter.

From his investigations at Millbank prison, Dr Baly came to the conclusion that the epidemics of dysentery there were due to a malaria arising from the soil; and Professor Maclean subsequently endorsed this opinion, and further attributed the poison to the decomposition of organic matter in the ground. In the meantime, however, the course of events at Millbank has proved conclusively that Dr Baly was wrong. In the year 1854 the prisoners ceased to be liable to dysentery; and during the next eighteen years (up to 1872) one death only occurred from that disease or from diarrhœa. Indeed, so far as I am aware, the immunity has continued down to the present time. Now, as Mr de Renzy has shown, one, and one only, change in its hygienic arrangements has coincided in time with this improvement in the sanitary state of the prison. Formerly, the water which the convicts drank was taken directly from the Thames as it ebbed and flowed beneath the walls. But on August 10th, 1854, the artesian well in Trafalgar Square was made the source of water supply to the prison; this has since continued. The change was effected in the middle of a cholera epidemic; six days afterwards the disease suddenly ceased. Enteric fever, too, no longer attacks the convicts, and the death-rate has declined to an extraordinary extent. It is, I think, impossible to avoid the conclusion that the exciting cause of dysentery in Millbank prison was the Thames water; and in all probability the noxious ingredient was derived from the sewage contained in it.

Another series of epidemics, which have been traced to a somewhat similar origin, occurred in the Cumberland and Westmoreland Asylum. In 1864, chiefly between May and August, twenty-six persons were attacked with dysentery, and in March, 1865, five others. For a long time Dr Clouston, the medical superintendent, was completely at a loss to account for the disease. It had often occurred to him that the cause might be connected with the distribution of the sewage of the asylum, which after being thrown into a large tank, was allowed to flow over a field about 300 yards distant. But it was not until August, 1864, that an offensive smell was noticed at the asylum during several hot and sultry evenings; and Dr Clouston then had the sewage carried away in a covered drain to a distance; from that time no fresh cases of dysentery occurred. An investigation was then made as to the exact meteorological conditions which had existed during the prevalence of the epidemic, and it was found that within a week before the day on which each patient fell ill there had always been either hot sultry evenings with no wind in the night, or northerly winds which blew from the direction of the field which was being irrigated. Male and female patients, too, were attacked at different times, according as the exact direction of the wind was such as to carry the sewage emanations either to one or to the other of the parts of the building which the two sexes severally occupied.

The probability that these observations pointed to the real cause of the dysentery was greatly increased by the fact that the five cases of dysentery in March, 1865, all occurred within a week after the sewage was again allowed to flow over the field, during one calm night, when the direction of the wind was towards the asylum. And I think the evidence may be said to have amounted to proof in the year 1868. There had then been two years' immunity from dysentery, and, the most approved precautions having been taken, it was determined to run the sewage over another field. Two months later six patients were attacked with dysentery and diarrhœa within a few days of each other; they were all in that part of the asylum nearest the field, and the wind had been blowing towards it continuously for eight days before the outbreak occurred. It must be mentioned that the subsoil was a stiff clay, through which water could not penetrate, and which therefore was entirely unfit for irrigation purposes.

So far as I know, these very interesting observations derive very little support from the recorded experience of military surgeons. But, on the other hand, this experience is not in any way adverse to them; and in time of war, as well as in tropical epidemics, the conditions are generally so complicated as to defy analysis. Dr Chevers, indeed, has expressed the belief that much of the dysentery (and cholera) occurring on board vessels in the port of Calcutta is caused by men drinking the water taken up in buckets over the ship's side, this water being loaded with excrementitious matters, and the products of putrefaction. And Heubner was told by several military surgeons, on whom reliance could be placed, that where many severe cases of dysentery were crowded together the disease was often spread by the latrines, ceasing when the proper precautions were taken with regard to them. Prof. Maclean says that in India, the barrack-rooms most exposed to the effluvia from latrines always furnish the largest number of dysenteric cases.

We must believe, then, that the cause of dysentery may be, and probably always is, the entrance into the body of some organic matter conveyed either by drinking-water or through the air. So far dysentery resembles enteric fever and cholera.

The next questions are whether, as in these diseases, the organic matter is a specific poison, and whether this poison undergoes a process of self-multiplication in the human body, so that the discharges possess special powers of infecting others with dysentery. Probably these questions cannot yet be finally answered. All observers, however, are agreed that the disease seldom or never passes from the sick to those who are attending on them. And Heubner lays stress on the fact that it has hardly ever been known to spread from the military to the civil population in time of war.

In Ziemssen's 'Handbuch,' however, dysentery is placed among the infective diseases, and Liebermeister explains that he regards it as belonging to the same class of maladies as cholera and enteric fever. He goes on to admit that it is a local disease, and that all its symptoms are dependent upon the intestinal inflammation; but, as he remarks, gonorrhœa and the soft chancre are *local*, and yet *infective* in the fullest sense of the term. Still the presumption is surely in favour of the view that no specific contagion is concerned in the production of a disease which is admitted to be a local inflammation; and this presumption is greatly strengthened by the fact that epidemic and sporadic dysentery cannot be separated by any strict boundary line. It has indeed been urged that between these two forms of the disease the same relation exists as between Asiatic and English cholera; and since the occurrence of a simple variety of cholera does not prevent the acceptance of the view that, when introduced from the East, this malady is due to a specific contagion, it is argued that the existence of sporadic dysentery is no bar to the infective theory of epidemic dysentery.

It would appear, too, that strong evidence in this direction has been afforded by an epidemic which occurred in Norway in 1859. Dysentery had not prevailed there for half a century, and as the population was scattered, and the ground very broken, there were unusual opportunities of tracking its course. The disease was studied with great care by Homan and Hertwig, and they believed that they established the fact of its spreading by a definite contagion, which was even conveyed by healthy persons from infected places to other spots where the disease had not before appeared. It does not seem that they traced the poison to any particular source, but the analogies of enteric fever and cholera evidently render it in the highest degree probable that, if there really is a contagion which multiplies itself in the human body, this reaches the outer world in the alvine evacuations.

With regard to the period of *incubation* in dysentery, Dr Clouston states that one of his patients fell ill within three days after first inhaling the poison; but others were attacked as long as three, four, or five days from the cessation of opportunities for its entrance into the body. Homan and Hertwig state that in Norway in 1859, the period of incubation was from two to eleven days.

Symptoms.—Before the characteristic features of dysentery develop themselves, there is generally a period in which the patient suffers from simple diarrhœa, with more or less griping pain in the abdomen. This may last from three to five days, or even a fortnight. He then becomes worse and perhaps he shivers, or at least complains of greatly increased malaise and weakness. The abdominal pain is more severe and paroxysmal. The desire to go to stool becomes more and more frequent, and, instead of abundant liquid fæces, he begins to pass only very small quantities at a time, with violent straining, and burning pain in the rectum. He is then said to suffer from "tenesmus," but although this term is universally employed there are hardly two writers who are agreed as to its exact signification. Some mean by it the peculiar sensation that there is "something in the rectum that wants to come away," others the straining, and others the burning pain; while others again include two or all in the connotation of the word. But as all these symptoms are constantly associated together it is really not a matter of importance whether tenesmus means one or another of them. So incessant may be the call for relief that the patient goes to stool twenty or thirty or even as many as two hundred times in the twenty-four hours. Or he may sit there for half an hour at a time, straining violently, but passing little or nothing. In a number of cases of dysentery occurring in camp, Heubner had the total daily evacuations collected. The quantity passed by each patient was found to be only from 28 oz. to 42 oz.

The evacuations in dysentery often contain no fæcal matter whatever. Small hard scybalous masses may be voided occasionally, but I think that all the most careful recent observers are agreed that these are comparatively seldom seen. Thus, the paradoxical expression of Stoll is justified, who described dysentery as *morbus alvum occludens.* But in some cases fluid fæcal matter is passed from time to time throughout the whole course of the disease, and this is said to be rather an unfavourable sign, as showing that the upper part of the large intestine, or even the small intestine, is involved. In most cases, however, the only result of the straining efforts of the patient is that he passes two or three drachms of gelatinous mucus, colourless, or more or less deeply stained with blood. Or there may be membranous shreds of mucus, and small clots, mixed with more or less of the fluid fæcal matter. Or, again, pure blood may come; or a thin reddish fluid, which has floating in it a number of little yellow or red masses, soft, and looking like pieces of sodden meat. This kind of discharge is often described as resembling the washings of meat and was formerly called *lotura carnea.* The masses suspended in it were formerly supposed to be cast-off portions of the intestinal mucous membrane, but it is now known that this is not the case. Dr Goodeve, in Calcutta, is, indeed, said to have found membranous sloughs from half to one inch or more in diameter, in the sediment obtained by washing the evacuations after the eighth to twelfth day of the disease. But, according to Heubner, it very rarely happens that masses are passed the nature of which can be recognised. Another dysenteric product is pus, which may occasionally be discharged pure and odourless, and just as if it had come from an ordinary abscess, but which is much more commonly mixed with fluid fæcal matter and blood. In some cases, again, a substance is voided which looks like frogs' spawn or boiled sago. This contains rounded bodies which were once thought to be sloughs derived from the solitary follicles. Heubner, however, says that they are too large for this, and that they consist of mucus. Lastly, the discharge may be a brownish-red, or blackish fluid, of a most horribly offensive odour; this is an indication that sloughing is going on in the intestine. It is to be noted that in all but the earliest stages of dysentery the matters passed from the bowel have an exceedingly disagreeable odour, which Parkes and others regard as peculiar to the disease, but which I think I have observed in cases of cancer of the rectum.

The abdomen is not at first distended, but it may become so as the disease advances. Tenderness may be altogether absent, or pressure over different parts of the large intestine may give more or less pain. In some cases one can obscurely feel an induration in the course of the bowel, and it has even been suggested that the extent to which the upper part of the colon is affected may be determined in this way.

Another symptom which is sometimes present in severe dysentery is strangury. It may be necessary to have the urine drawn off by a catheter.

The most marked general symptom is the great prostration. The patient not uncommonly faints while he is at stool. Anæmia is very rapidly produced; the face assumes a pale, waxy look. The febrile disturbance in this disease is generally slight. Even in severe cases the temperature may be normal or rather below normal, but sometimes it rises to 100° or 101°. The pulse is not much quickened, except in certain epidemics in which the disease was formerly characterised as "inflammatory dysentery." The patient, however, often makes great complaint of thirst. The appetite is not always lost, but griping pain is often brought on by food so that he is unwilling to take any but the blandest nourishment. Cold liquids frequently have the same effect, and therefore it is well that what the patient drinks should be lukewarm.

Diagnosis.—A question of great importance is whether dysentery is always attended by the very striking train of symptoms which I have been describing. Now, writers mention that some patients pass no blood, and I think it is said that certain epidemics are characterised by the absence of this symptom. Again, it is well known that tenesmus and the other symptoms which are constantly associated with it depend upon disease of the lower part of the large intestine, and that they are absent when the inflammation is limited to the cæcum and the upper part of the colon. Dr Clouston says that in the epidemic which he observed at the asylum near Carlisle, some patients experienced scarcely any pain throughout the whole course of the disease, and at first, having no fever or want of appetite, they refused to believe that they were ill, although they were passing glairy mucus mixed with blood. But I do not find that any writer speaks of dysentery as being ever latent when the disease is epidemic. I have, however, seen at least two well-marked instances of fatal sporadic dysentery occurring in London, in each of which there was very extensive diffused ulceration of the large intestine, the presence of which had been entirely unsuspected during life. Each patient died in Guy's Hospital, the one of an enormous hepatic abscess, the other of a large abscess in the left iliac fossa, resulting from extension of ulceration from the bowel into the retro-peritoneal connective tissue. Dr Dickinson has recorded a similar case, that of a woman who died in St George's Hospital of abscess of the liver, and who, during the week which she passed as an inmate of the hospital, had constipation so as to require aperient medicines. She had not allowed that she had ever had any looseness of the bowels, but after death the upper part of the large intestine was in a state of ragged ulceration.

Again, in at least three cases of acute dysentery that have proved fatal in Guy's Hospital the disease was supposed during life to be enteric fever; the characteristic symptoms were either absent or passed under the name of diarrhœa. It is worthy of inquiry whether a similar latency or obscurity of symptoms may not sometimes occur in epidemic dysentery, both in India or in temperate climates. We shall hereafter see that the question is one of considerable interest in reference to the causation of abscess of the liver.

On the other hand, when all the symptoms of dysentery are present, the diagnosis should, I think, be made with more caution than would appear necessary from the statements of writers, who speak of it as presenting no difficulty whatever.

In young persons, and particularly in male children, one must always bear in mind that an intussusception causes very similar symptoms, although they no doubt begin in a different way. In this country I have more than once seen a case of intussusception mistaken for one of dysentery, and I cannot doubt that this very serious error must often have been committed in those parts of the world in which the latter disease, being epidemic, is likely to have its presence taken for granted.

On the other hand, in persons more advanced in years it very frequently happens in England that cancer of the rectum is overlooked, and that its symptoms are attributed to dysentery. I know of so many examples of this that I would almost lay it down as a rule that a supposed case of chronic dysentery in a middle-aged or old person, who has not been out of England, is really one of local disease of the rectum, most probably cancer. A digital examination generally at once clears up all doubt as to the nature of the disease; and very likely it would be possible in most cases to determine by careful investigation that the blood which is passed during defæcation is not intimately blended with the motion but comes before or after it.

Progress and event.—The course taken by dysentery varies much in different epidemics, as well as in individual cases; we shall hereafter see that it may to a very great extent be modified by treatment. In favourable cases the symptoms quickly begin to decline. First the pain and tenesmus pass off, then some of the evacuations begin to contain fæcal matter. Formed fæces may for a time be passed alternately with blood and mucus.

An occasional sequela is an affection which somewhat resembles acute rheumatism. According to Homan and Hertwig, it attacks several joints at the same time, but these do not become very tender, nor is there much swelling. The febrile disturbance is moderate, and the affection subsides in three or four weeks. Parotitis is also said to occur sometimes during convalescence from dysentery.

In cases which are about to terminate fatally the prostration passes into collapse. The features become sunken. The body is covered with cold sweat, and exhales a fœtid odour. The urine may be suppressed. The tongue and lips are covered with sordes. Hiccup sets in, and a painful sense of constriction in the epigastrium is complained of. The fæces are passed involuntarily. The anus and penis become excoriated, and the lower part of the rectum is often prolapsed. Bedsores are formed if the patient should live long enough. Consciousness is often retained to the end, but in some cases the mind wanders. And during the last few hours all pain may cease, so that the patient fancies he is doing well, and his delirium assumes pleasing forms. Before death the temperature of the body often rises considerably.

Prognosis.—With regard to the grounds on which a forecast must be based in a case of dysentery, I have little to say that has not been implied in preceding paragraphs. The severity of the disease is proportional to the extent and intensity of the local inflammation, but the latter are nevertheless to be measured during life by the gravity of the constitutional rather than of the local symptoms. Tenesmus and pain may be slight or even absent, and yet the patient may be in very great danger. His sufferings may be extreme; and yet there may be no grounds for alarm, the affection being limited to the rectum. But the appearance of his countenance, the state of the circulation, the presence or absence of symptoms of collapse, generally enable a correct judgment to be formed as to the probable issue of the case. The rate of mortality varies greatly in different epidemics. In Dr Clouston's Asylum it was 64·5 per cent. This is enormous, for even in Western Africa the proportion of deaths to admissions appears from a table drawn up by Sir Alexander Tulloch to be only 14·2 per cent; and the death-rate was higher than in any other of the intertropical stations for British troops.

In epidemic dysentery the prognosis must also be greatly influenced by the presence of scurvy as a complication. This is exceedingly frequent, so that many observers speak of scorbutic dysentery as a distinct variety of the disease. But I shall not describe the symptoms of such a form in detail, because they are in fact made up of a combination of those which belong to the two maladies separately. Only it may be worthy of mention that when scurvy is present the course of the disease is slower than in other equally severe cases in which it is absent. The patient almost always lives three weeks, and often lives as long as three months.

Even the most severe form of uncomplicated dysentery is seldom rapidly fatal. Death rarely takes place within the first week, or before the ninth or tenth day. At Guy's Hospital, however, some years ago a case occurred in a child who died after five days' illness. She was in the hospital for chorea when she was attacked, and it was a question whether the sulphate of zinc which she was taking could have been concerned in causing the intestinal inflammation. The symptoms were so severe that the case was mistaken for one of Asiatic cholera, that disease being prevalent at the time.

In the later stages of dysentery, perforation of the intestines sometimes occurs, setting up fatal peritonitis. I find notes of one case, in which this was observed in a patient at Guy's Hospital. He was in a surgical ward for disease of the knee, when in the month of August he was attacked with "severe diarrhœa." This continued, and he died at the end of a fortnight. The large intestine was ulcerated and sloughing in its whole length, and at one spot the transverse colon was perforated. But in three other cases of sporadic dysentery at Guy's Hospital there has been acute peritonitis without any perforation being discovered to account for it. I do not know that this form of serous inflammation has hitherto been observed in epidemics of the disease.

In other cases, as I have already mentioned, acute dysentery passes into a *chronic form* of the disease, which may last for months or even years. I do not find it stated by writers of experience that chronic dysentery may also arise without being preceded by any acute stage; but, if not, this disease is an exception to a rule which is, I believe, observed in the case of all other maladies. In chronic dysentery the discharges still have to some extent the peculiar odour. They are for the most part liquid, but they vary in character from day to day. Sometimes they may contain tolerably natural fæcal matter, at other times they consist of a blood-stained mucus or serous fluid. The patient does not gain flesh and he may become more and more emaciated. The appetite is generally capricious. The tongue is red and glazed. Abscess of the liver not infrequently forms in such cases; or death may arise from pneumonia, phthisis, Bright's disease or lardaceous disease of the viscera; or again a fæcal abscess may make its appearance in the iliac fossa, or elsewhere. Peritonitis from perforation may also occur even at this period. Chronic dysentery is by most writers regarded as an exceedingly intractable disease and one which generally ultimately proves fatal, the patient dying by exhaustion, with febrile symptoms of hectic type, even if no complication should arise to cut short his existence. Dr Ward, however, says from his experience at the "Dreadnought" that in the majority of cases recovery at length takes place, if they are judiciously treated. Even then, however, the bowels often remain irritable and easily disturbed for a very long period afterwards, it may be for the rest of the patient's life.

Before entering on the treatment of dysentery we may ask whether any *prophylactic* measures can be adopted when the disease prevails, to protect those who have hitherto escaped. The answer to this question is that all general hygienic measures must be strictly attended to, overcrowding of men together being particularly avoided. As a precaution it is proper that the evacuations of those who are sick should be disinfected with carbolic acid, and removed without delay, although, as we have seen, it has not yet been proved that they contain a specific poison. Those who are still well must clothe warmly and be careful to avoid chills; they should eat and drink moderately, taking no strong wine or spirits, and they should not allow the bowels to become constipated. It is not necessary, as a matter of course, that ripe fruit should be excluded from the dietary.

Treatment.—The therapeutics of dysentery must be based upon a knowledge of the natural course of the disease. Now, Dr Austin Flint, of New York, some years ago observed ten cases in succession in which no medicine was administered except a little tincture of cinchona as a placebo. And he found that the mean duration was eleven and one fifth days; the most protracted case lasting twenty-one days, the shortest six days. Or, reckoning from the first dysenteric evacuation (instead of from the commencement of illness), he obtained a mean duration of eight and a half days, the maximum and minimum being twelve days and five days respectively. Previously the same observer had analysed forty-nine cases, which had been treated, some with calomel and opium, some with opium alone, some with castor-oil, and others with astringents. The mean duration was almost exactly the same in these cases as in those in which the disease ran its natural course.

These particular figures are evidently applicable only to dysentery as it occurs in the city of New York, and in strictness only to the actual period within which the observations were made. It is clear that Dr Flint's cases were mild ones; one point on which he lays stress is that the complaint showed no tendency to become chronic and that relapses never occurred, although he allowed his patients to eat solid food as soon as they chose to do so. Very different is Prof. Maclean's account of dysentery as he saw it in India. "Speaking from large experience," he says, "I affirm that complete restoration to health, by the unaided efforts of nature, is of extremely rare occurrence; the disease either destroys the patient or it passes into a chronic form."

But it would seem that Dr Flint's observations fairly establish the uselessness of the various remedies employed in his earlier series of cases. And it is therefore a gratifying circumstance that we have for dysentery another medicine of which he does not seem to have made trial, but which is proved by the concurrent testimony of a large number of observers to possess the power of cutting short the disease and even of curing cases that would otherwise have terminated fatally. That medicine is ipecacuanha. The root of this plant was first employed as a remedy for dysentery in Brazil, where it is indigenous. Towards the end of the seventeenth century it was introduced into France as the *radix antidysenterica*, and being successfully given to the Dauphin it acquired a great reputation. In India it was used even before mercury came into vogue, and of late years it has become the staple remedy. The method of administering it which is now generally adopted was introduced by Surgeon Docker, of the 7th Royal Fusiliers. Prof. Maclean gives the following directions.

The patient having been put to bed, twenty-five to thirty grains of powdered ipecacuanha are given to him in as small a quantity of fluid as possible; a little syrup of orange peel covers the taste as well as anything. Some surgeons think it of importance that thirty minims of tinct. opii should be given half an hour before, in order to make the stomach tolerant of the ipecacuanha; but Prof. Maclean says that he has seen the latter drug well borne without any such precaution having been taken. After the dose, the patient should keep perfectly still, and abstain from drinking for at least three hours. If thirsty, he may suck a little ice, or have a teaspoonful of cold water at a time. Under this management he seldom complains of excessive nausea, and vomiting rarely sets in within two hours. A poultice is in the meantime placed over the abdomen, or a piece of spongiopiline wrung out of hot water, with a little turpentine sprinkled over it. Afterwards some bland nourishment is given. In from eight to ten hours, according to the urgency of the symptoms and the effect of the first dose, the ipecacuanha is repeated, its quantity being somewhat reduced, but with the same precautions as before.

"All who have had opportunities of trying this mode of treating dysentery," says Prof. Maclean, "can bear testimony to the surprising effects that often follow the administration of one or two doses of ipecacuanha. The tormina and tenesmus subside, the motions quickly become purulent, blood and slime disappear, and often after profuse action of the skin the patient falls into a tranquil sleep, and awakens refreshed." The remedy may, however, require to be continued in diminished doses for some days; and even after the stools have regained a healthy appearance it is well to administer ten or twelve grains at bedtime for a night or two.

Even when the powers of life are very low, this remedy may sometimes be given with safety and success. Prof. Maclean mentions the case of a lady who landed at Calcutta, having come from Madras, and who was so exhausted that her voice was scarcely audible. With some misgiving he gave twenty-grain doses at intervals of eight hours; after the third dose she was out of danger.

When severe vomiting follows the administration of ipecacuanha Prof. Maclean says that coexistent liver disease may be suspected, or complication with malarious fever. In the latter case he advises that quinine in ample doses should be alternated with the ipecacuanha.

In mild cases, this writer recommends that the treatment should be commenced with a hot bath, which must be brought to the patient's bedside. He is to be kept in it until he feels faint, and after being rapidly but carefully dried he is to be put to bed, and to have a dose of fifteen to twenty grains of ipecacuanha. In some cases a few drachms of castor-oil, with a little tincture of opium may be afterwards prescribed. A turpentine stupe should be applied to the abdomen, and repeated from time to time. According to Heubner enemata of starch and opium often give great relief to the tenesmus in these mild cases.

The action of ipecacuanha in acute dysentery is, I think, as yet unexplained; and it must be regarded as a specific. Professor Maclean supposes that it is an "evacuant," increasing the secretions of the alimentary canal. Its administration renders unnecessary the use of castor-oil, tamarinds, rhubarb, or the sulphates of potass or soda, which are recommended by different French and German writers. It is curious that even Heubner, writing in 1874, seems to know scarcely anything of the value of ipecacuanha, except as an emetic. But he has heard that it was used by some English surgeons with good results in the war between France and Germany. Calomel has been definitely abandoned in the treatment of dysentery. Opium is believed by all experienced observers to be injurious, if systematically given. Venesection has been laid aside, as tending to exhaust the patient's strength to no purpose.

The value of this method of treating dysentery is attested not only by individual experience, but by statistical results, which are so striking that one is inclined to place an unusual amount of dependence upon them. In Bengal, under "the old system," the average mortality among Europeans during forty-two years was 88·2 per thousand; in 1860, under ipecacuanha it was 28·87 per thousand. In Madras, the corresponding numbers were respectively 71 and 13·5. Lastly, Surgeon Mee, at Madras, treated sixty-eight cases from the 44th Regiment "in the ordinary way," with a mortality of 6 (or 88 per thousand); afterwards he treated fifty-nine cases with large doses of ipecacuanha, and these all recovered. It is also asserted by the advocates of this remedy that the number of chronic cases of dysentery diminishes year by year as its administration becomes more and more general; and the development of hepatic abscess, as a complication, becomes less frequent.

Against these statements it is proper to set Dr Clouston's experience in the epidemic at the Cumberland and Westmoreland Asylum. He found the ipecacuanha treatment useless, even if (as he appears to think) it did not take away the last chance from one or two of the patients by causing vomiting that could not be stopped and prostration that was never rallied from. But, as I have already stated, this epidemic was one of a type far more severe than ordinary tropical dysentery. And it is a cardinal rule,—although one which is too apt to be forgotten,—that in all the worst cases of a disease a medicine may fail to produce any appreciable benefit, and yet it may be capable of curing those which tend but a little less surely towards a fatal termination.

In such severe and malignant forms of dysentery, Professor Maclean recommends the solution of pernitrate of iron; he says that he has sometimes prescribed ten drops every hour with advantage. The patient must of course be sustained with nourishment, and stimulants may often be freely given. Dr Clouston found that milk boiled with a little flour, and allowed to cool, was taken more readily than anything else, and kept up the patient's strength. He also gave strong beef tea, jelly and eggs, and wine and water *ad libitum*.

In *chronic dysentery* an essential part of the treatment is removal to a better climate. In India it sometimes suffices to send the patient to the seaside; but more often a voyage to Europe is necessary. Persons invalided home on account of chronic tropical dysentery are occasionally seen in the London hospitals. In such cases rest in bed is a very important part of the treatment. Dr Ward has especially insisted on this fact, and on the necessity that the diet should be very carefully restricted, milk and farinaceous food being the best that can be given. His experience at the "Dreadnought" has led him to believe that ipecacuanha is useless in such cases. But Prof. Maclean says that exacerbations of a subacute character are of frequent occurrence in chronic dysentery, and that at Netley he has often given the ipecacuanha in suitable doses with the happiest effect. My own experience at Guy's Hospital has certainly been that this medicine is of great value even when dysentery has been brought home from the East. I remember at least one instance in which it did good, even in out-patient practice. H. P—, aged thirty-five, was in Barbadoes several years. Attended as out-patient for the first time on May 20th, with chronic dysentery of three years' duration. He states that he has been accustomed to go to stool five or six times daily, and has passed considerable quantities of blood at different times. Ordered Pulv. Ipecac. gr. ij, in pill, three times daily. On May 27th much better. Since Friday last, he has only had one motion daily, and has seen no blood.

Dr Ward's patients were probably sailors, whose food on board ship had been such as must necessarily aggravate the disease to the highest point; and it is not surprising that they should derive so much benefit from rest in bed, and a milk diet, that the effects of medicines were thrown into the background. Even when a person affected with dysentery is sent home as an invalid, with nothing to do but to take care of himself, the disease is very apt to become worse on board ship, unless special care is taken that he has proper food and puts on warmer clothing as he passes into a more temperate climate.

In some cases of chronic dysentery, astringents are of great value. Professor Maclean recommends the solution of pernitrate of iron, which, besides checking the discharges, removes anæmia. Krameria, catechu, hæmatoxylum, even tannic and gallic acids, may each be useful in turn. I have seen marked benefit result from the administration of drachm doses of the extract of Indian bael. This hardly accords with the opinion of Professor Maclean that even the fresh bael fruit is efficacious in dysentery only when a tendency to scorbutus is also present. Acetate of lead and sulphate of copper are sometimes useful. Dr Ward mentions one case in which enemata of nitrate of silver (gr. iv ad aq. ℥ij) were repeated at night-time with good effects.

Intestinal Obstruction.—We have now to pass to the consideration of a group of affections which differ widely from one another in their anatomical characters, but which agree in this, that they tend to cause mechanical obstruction of the contents of the bowels.

Invagination.—Of these affections there is no one more interesting, or more important, than that which is termed intussusception or invagination, when one part of the bowel passes into another part, just as the finger of a glove can be made to slip into itself. The direction in which this occurs is invariably downwards (or "forwards"); *i. e.* the portion of the gut which receives the other portion is always on its anal side.

A moment's consideration will show that an intussusception must consist of three parts, or, as they are often called, "layers." Of these we may term the outermost the "receiving," the middle the "returning," and the innermost the "entering" layer. The returning layer, unlike the others, has its mucous outside its serous coat; it is in fact turned inside out. The bend which connects the receiving and the returning layers is situated at the upper part, and its convexity is formed by the peritoneal coat of the intestine; that which connects the returning and the entering layers is at the lowest point of the intussusception; and the mucous membrane covers it.

Probably almost all portions of the bowel are liable to intussusception. Sometimes one part of the small intestine enters another part, or one part of the colon another part of it. A case has occurred at Guy's Hospital in which the rectum with just the lower end of the sigmoid flexure passed down through the anus; there was some difficulty in distinguishing it from a mere prolapsus of the mucous membrane. There is, however, one point at which invagination occurs much oftener than anywhere else, namely, at the junction of the small with the large intestine. Such cases are generally distinguished as *ileo-cæcal* intussusceptions. It is commonly supposed that they arise by a gradual protrusion of the ileum through the ileo-cæcal valve into the cæcum. But in reality this rarely occurs; I do not know that more than four or five instances of it have been recorded. Almost always the cæcum passes with the ileum into the colon.

An invagination at its commencement affects only a very small part of the bowel. Gradually more and more of this is involved, and always by the inclusion of one part after another of what had been the receiving layer. Thus the upper bend of the intussusception is constantly shifting, but the lower bend, on the contrary, remains stationary from beginning to end. In an ileo-cæcal case, for instance, the entering layer is the ileum; the cæcum usually constitutes both the returning and the receiving layers; the lower bend is situated exactly at the ileo-cæcal valve. As the affection advances, the whole of the cæcum, the ascending, transverse, and descending colon may become included; but the ileo-cæcal valve always remains at the lowest point of the mass. Extraordinary as it appears, an invagination of this kind may pass through the anus, and even project for some inches; and the valve may actually be seen and felt in this position, as well as the orifice leading into the vermiform appendix. The peritoneal layers fixing the intestine, and the vessels which pass to it, must indeed be stretched to an extent which it is difficult to conceive; but the protrusion of an ileo-cæcal intussusception from the rectum has repeatedly been observed, and may be regarded as the natural course of the affection. On the other hand, when the small intestine is invaginated, the mesentery tethers it much more closely. It is drawn in, and forming a wedge-shaped mass on one side of the gut, pulls on it and drags its lower end to one side, or makes it assume a contorted or spiral form.

The next step is that the circulation of blood in the invaginated mass is interfered with. Sometimes, it seems, the veins alone are compressed; blood can then no longer return from the affected part of the bowel, which becomes enormously swollen, with hæmorrhage and exudation of serum into its tissues. This is particularly apt to occur in ileo-cæcal intussusceptions; but it is often delayed until an advanced period of the case. The swelling is always more marked at the lower bend than anywhere else. Dr Moxon mentions an instance in which the coats of the bowel were three quarters of an inch thick in this position. The included part of the mesentery likewise becomes dark and thickened by effused blood.

In other cases the influx of blood through the arteries appears to be arrested as well as its escape by the veins. This takes place especially in intussusception of the small intestine, in consequence of the comparatively narrow diameter of the receiving layer in that form of the disease. The inevitable result is the occurrence of gangrene of the invaginated mass. And, strange as it may appear, this does not necessarily lead to the death of the patient. The sloughing part of the bowel may be cast off, and may pass down the large intestine, and be discharged per rectum. It generally appears as a single tube, with its mucous surface outwards; this perhaps includes both the entering and the returning layers, the former having undergone inversion during the process of detachment; or it may be that the entering layer is cast off separately in the form of soft shreds, so that the inverted mass is constituted by what had been the returning layer only. Some years ago I examined a specimen of this kind which had been sent up to Guy's Hospital by Mr Higginbotham, of Bruton; it consisted of twelve inches of intestine, and within its channel lay the appendix vermiformis, which opened on to its outer or mucous surface. Only an inch of it was small intestine. Consequently, if the intussusception was of the ordinary ileo-cæcal variety, the cast-off mass must have been derived mainly from the returning layer, the entering layer having doubtless broken down and been discharged separately. Several instances have been recorded in which portions of bowel from twenty to forty inches long have been shed; and in one extraordinary case related by Carswell eight distinct pieces, amounting altogether to fully twelve inches in length, were passed per anum at considerable intervals of time. In different cases there are great variations in the extent of the inflammation that occurs at the line of separation, which of course corresponds with what was the upper bend of the intussusception. In a case which Dr Hare brought before the Pathological Society in 1862, and in which the patient died of phthisis three months after passing some inches of bowel, the line of union could only just be detected on the mucous surface by its shining glazed appearance. Even on the serous surface there was only what is described as "considerable puckering," but below the cicatrix a small pouch existed into which projected a curious little hollow cylinder, evidently a relic of the invaginated part. In other instances, however, inflammatory products have been formed in large quantity at the line of separation; and thus the new channel has been reduced to a very small calibre. Or suppuration has occurred at the same spot, and an abscess has developed itself containing pus alone, or pus mixed with fæcal matter. Cases of this kind probably always terminate fatally. And even when the expulsion of the cast-off bowel is followed by the recovery of the patient, there is always a risk that the cicatrix may gradually contract. Dr Moxon has twice seen a fatal annular stricture of the ileum, with puckering of the mesentery, which he believed to have arisen from a former intussusception.

Ætiology.—The causes of intussusception are imperfectly understood, but there is much that is suggestive in what is known. It may occur at any age. In adults, however, it is infinitely more rare than might be supposed from the comparatively numerous cases that Hutchinson, Peacock, and others have collected from different medical works. Dr Wilks used to say that he had only seen one case in a grown-up person. On the other hand, in young children and infants the disease is very frequent; and probably very many cases are overlooked, so that it really occurs oftener than would appear from the published statistics. It is a very curious circumstance that, even among infants, males are far more liable to intussusception than females. Rilliet and Barthez met with twenty-two cases in boys to only three cases in girls. I do not know what explanation of this it is possible to give, unless one ascribes it to the greater restlessness and activity of male, as compared with female, children. There is reason to believe that sudden movements of the body may cause intussusception. Rilliet and Barthez mention two instances, in each of which it came on suddenly in a child who was being jumped in the father's arms. Violent muscular efforts to raise a burden, and falling upon a seat, have also severally been assigned as causes of it; and likewise direct injuries, such as the passing of a wheel over the abdomen, a kick from a horse or a severe punch in the belly.

It probably suffices for the production of an intussusception that the contraction of the transverse muscular fibres of one portion of the bowel should lengthen it and push it into the interior of the succeeding portion. Everyone who is accustomed to make post-mortem examinations knows that in children, particularly after death from cerebral disease, it is common to find two or three, or even several, short intussusceptions at different parts of the small intestine. The affected parts are not reddened in such cases, and there have been no intestinal symptoms; hence some writers have supposed that the invaginations have arisen after death as a result of rigor mortis. But it appears more probable that they really occurred during life, and that had life been prolonged they would have undergone spontaneous reduction. One distinction between such intussusceptions and those which give rise to symptoms is that (so far as I know) the former never occur at the junction of the ileum with the cæcum, which we have seen to be the favourite seat of the latter. The special liability of this part of the bowel to the disease is probably the combined result of two different circumstances; one, that the axis of the large bowel is nearly at right angles with that of the small intestine; the other, that the cæcum is much more fixed as well as much larger than the ileum. I have met with references to two cases, in each of which the occurrence of an invagination of the ileum appeared to have been favoured by the presence of cancerous induration of the cæcum; this may be supposed to have kept it more widely open than in its normal condition. On the other hand, many instances have been recorded in which the starting-point of an intussusception has been a polypus hanging from the mucous membrane. This seems to have been caught by the contraction of the intestine below, and to have dragged downward in its turn the part of the bowel to which it was attached. Dr Moxon met with a case in which an intussusception appeared to have been caused in a similar way by a diverticulum of the ileum; this must itself have been first inverted into the gut. The presence of the ascaris lumbricoides in cases of intussusception have been noticed by several observers, and it seems not impossible that a worm might have its body grasped by the peristaltic movements of the intestine, so as to invert the part to which its head was attached.

Symptoms.—Intussusception was formerly supposed to be characterised by the same symptoms as the various forms of intestinal obstruction, with but slight modification, and was, indeed, often spoken of as the typical and ordinary cause of that condition. But the fact is that, as a rule, the effects of invagination of the bowel are altogether different, and in the progress of some cases two distinct periods can be recognised, each attended with its own symptoms.

In the *first period* the intestinal contents pass freely through the intussuscepted part. The bowels may act every day and the evacuations are natural. The chief complaint is of a paroxysmal *pain* in the abdomen, the first sudden onset of which forms in fact the starting-point of the case. This pain is generally referred to the neighbourhood of the umbilicus. Dr Brinton says that it is sometimes distinctly recognised as of a straining or tearing character. I have not myself observed that it differs from the pain which may arise in any other acute affection of the bowels. It is often extremely violent, so that the patient rolls about the bed or the floor of the room in agony. It is sometimes accompanied by vomiting, but rigors seldom occur. It passes off and may return only after a considerable interval. In a case that came under my own observation, the patient, a boy five years old, had for four months only two or three attacks of pain during each twenty-four hours; between them he appeared perfectly well.

There is, however, another symptom of intussusception which can almost always be detected by manipulation of the abdomen. I refer to the presence of a *tumour*. This is in fact a most important feature of the disease, even from its very commencement. To detect it one may sometimes have to place the patient under chloroform so as to relax the abdominal walls, and if there be a great quantity of fat in them and in the mesentery, the examination may fail to yield a definite result. But with these limitations I think that, as Mr Hutchinson has said, all doubt as to the existence of an intussusception may generally be cleared up by thorough palpation of the abdomen. The tumour which occurs in this disease is felt as a sausage-like mass of greater or less length. The note yielded by percussion over its surface may be dull, or partially resonant, or scarcely distinguishable from that given by other parts of the abdomen. Its seat varies with the part of the bowel which is concerned. When this is the small intestine alone, Dr Brinton says that the tumour may occupy almost any region of the abdomen, but it is more commonly found in the hypogastric and right iliac regions than elsewhere. In the ileo-cæcal variety it originally occupies the right iliac fossa or its immediate neighbourhood. As more and more of the intestine becomes involved, the tumour in this form of the disease gradually changes its position. It moves across the abdomen, either at the level of the umbilicus or a little higher; having reached the left side it passes downwards into the left iliac fossa, and ultimately into the pelvis. Dr Brinton says that it often forms an elongated mass, which lies horizontally just above the pubes. Another peculiarity about this kind of tumour is that its size and form are in many cases liable to frequent changes. It may be hardly perceptible when one first places one's hand upon the abdomen, and may harden and become prominent under manipulation, particularly if a paroxysm of pain should come on. I have met with one instance in which so positive a diagnosis was founded upon the presence of two symptoms only—pain and tumour—that I got my colleague, Mr Howse, to open the patient's abdomen and to search for and pull out the intussusception. The case is recorded in the 'Medico-Chirurgical Transactions' for 1876. The patient made a good recovery.

As the disease advances, exploration of the rectum affords further aid in diagnosis. The end of the invaginated intestine can often be felt with the finger, and ultimately it may even protrude from the anus. Before this occurs, however, fresh symptoms commonly develop themselves, belonging to the *second stage* of the disease. I have already remarked that the veins of the intussuscepted part of the bowel become obstructed, and that it consequently becomes intensely congested and swollen. Another result of this change is that *hæmorrhage* occurs from its surface, and that blood is discharged per anum, or a mixture of mucus and blood. Some tenesmus may also occur if the affection extends into the lower part of the large bowel. Or blood may be vomited when the small intestine near the stomach is the seat of the disease. At the same time or somewhat later the passage of fæcal matter through the invaginated part is obstructed. *Sickness* sets in, there are no longer any remissions in the pain, the vomited matters become stercoraceous, the abdomen is enormously distended, the patient becomes collapsed, and before long he expires. In the case I have already referred to of a boy who for four months had no symptoms but paroxysmal pain and tumour, death occurred within three or four days from the time when he first began to have tenesmus and to pass blood and mucus. And in infants such symptoms generally set in at the very commencement of the disease, which commonly proves fatal by the second or third day.

It has generally been supposed that the cases attended with these symptoms are those in which the invaginated mass is apt to slough away and to be discharged per anum, but Mr Hutchinson has shown that in chronic cases of intussusception this very rarely occurs. And I have already pointed out that the changes which lead to the gangrene and shedding of the whole of the invaginated part of the bowel involve the complete arrest of the circulation of blood in it. Consequently, one would not expect that hæmorrhage from the bowels should occur while these changes are in progress, although it is true that the ulceration at the neck of the intussusception may lead to hæmorrhage at the time when the sloughing part is being detached; and I find that out of twenty cases collected by Dr Peacock, in all of which the invaginated parts were shed and passed per anum, there was only one in which bleeding is said to have occurred, and in that one it ceased twelve or fourteen days before the expulsion of the gangrenous mass. In fact the symptoms of those cases in which the affected part of the bowel sloughs can seldom be clearly divided into two stages, and very often they are undistinguishable from those of other forms of intestinal obstruction, until the portion of gangrenous intestine is unexpectedly voided from the rectum.

Among those cases which present the more characteristic symptoms of intussusception the duration of the first period varies greatly. I have myself seen a case in which it lasted four months, and similar instances have been recorded by others. A patient of Dr Brinton's died of cancer of lungs during this stage of an intussusception, which had lasted four months and a half, and it remained uncertain whether the abdominal affection had any share in determining the time of the man's death. Such protracted cases appear always to be examples of the ileo-cæcal variety of the affection. On the other hand, many cases, even of this variety, pass into the second stage from their very commencement; and when the small intestine is alone concerned, the disease appears generally to take this course. Sometimes hæmorrhage and the other symptoms belonging to the second period set in and afterwards subside. Mr Sydney Jones met with a case in which they lasted for three days and then passed off, returning again seventeen days later and then leading to a fatal termination.

Dr Brinton says that when the invaginated mass sloughs and is discharged per anum, the date at which it commonly separates is the eighth day, in cases limited to the small intestine; and that that on which it is expelled is the tenth day. In cases belonging to the ileo-cæcal variety the corresponding dates are, according to him, the fifteenth and twenty-second days respectively. But to these last figures I attach very little value, because of the variable duration of the first stage in ileo-cæcal intussusceptions.

The *diagnosis* of intussusception may either be perfectly easy, or exceedingly difficult. It should be laid down as a rule that the abdomen is always to be carefully explored, by the hand laid upon its surface, whenever a patient (particularly a child) complains of paroxysmal pain there, recurring without obvious cause. A tumour may perhaps be discovered, the form of which, and its seat, would be consistent with its being an intussusception. The alternative diagnosis generally is that the tumour is an impacted mass of fæces. An enema (repeated, if necessary) generally solves all doubt upon this score; or the suspicion may be confirmed by one's finding the tumour harden under manipulation, or detecting peristaltic movements in it. I remember one instance in which a rounded swelling, of somewhat cylindrical form, and only partially dull on percussion, was felt crossing the abdomen just above the umbilicus. At first I thought that the disease might be intussusception. But the fixity and unchangeability of the tumour, and the absence of other symptoms, soon showed that it was not. The man died some months afterwards, and the case proved to be one of tubercular peritonitis, the mass that had been felt being the omentum indurated with caseous tubercular matter.

In those cases which are attended at an early stage with hæmorrhage from the bowels, the danger is that one should mistake the disease for dysentery. All doubt may then sometimes be removed by digital examination of the rectum. But even when there is protrusion of the invaginated mass from the anus, it has sometimes been taken for a mere prolapsus. This occurred in a case which has been recorded by Mr Hutchinson; a practitioner, called in to see the patient, returned the bowel into the rectum, and fitted a cork pad to the anus to prevent its coming down again.

Treatment of intussusception has for its object the replacement of the bowel in its natural position. This may be effected by several different methods. In at least two cases introduction of a gum-elastic bougie into the rectum has succeeded, but it is applicable only when the invaginated mass lies in the rectum, and probably only when the part of the intestine concerned is limited to the lower part of the large intestine. The copious injection of warm water, again, has been known to cure the disease. But a much more effectual measure than either of these appears to be inflation of the intestine with air. This procedure was many years ago (1838) recommended by Mr Gorham in the 'Guy's Hospital Reports' (1st series, vol. iii, p. 345); and he quoted three cases which had been successfully treated in this way in America. Within the last few years it has been employed several times, and sometimes with the result of completely curing the disease. More often, I think, its success has been partial. The tumour has been reduced in size; or it has changed its position, returning towards the seat which it had occupied at an earlier period; or it has been made to disappear for a time, and all the other symptoms have subsided, but only to recur a few days later, with a fatal termination. The method of injecting air into the bowel is very simple.

An ordinary pair of bellows are employed. Round the pipe, at a little distance from the nozzle, a plug of lint is firmly fixed with adhesive plaister. The pipe is then introduced into the rectum, the plug being kept firmly fixed against the anus. Air is then forced in until the abdomen becomes tense, or until the patient can no longer bear the pain. Sometimes it has appeared to be advantageous to place the patient head downwards while either air or water is being injected into the bowel; and it is said that the inhalation of chloroform may be serviceable. Care must be taken that too much force is not used, for in the case of an infant, aged five months, which was treated by inflation in Guy's Hospital in 1873, the bowel gave way and a large quantity of air passed into the peritoneal cavity. Some of it was let out through a puncture in the abdominal walls, but the child did not rally and died in a few hours. And I have heard of another instance in which the serous covering in the bowel was cracked in several places and the muscular fibres torn; in that case both air and water were injected.

It is easy to see that air, as a highly compressible fluid, is far more likely to effect the reduction of an invaginated mass than an incompressible fluid, like water. And I think that inflation should be practised in every case of intussusception in which the diagnosis is made at a sufficiently early period. It is indeed evident that if the process of sloughing of the included bowel has once set in, the procedure would not only be useless but would take away the last chance of the patient's recovery. But as I have already remarked, there are no very definite symptoms which indicate that the invaginated mass is passing into a state of gangrene. Only, as Mr Hutchinson has suggested, the fact that in a particular case the tumour is advancing lower and lower in the large intestine is always proof that this is not the case, and that the upper bend is not yet fixed.

If it should be decided that such methods of treatment are inapplicable, the patient must be kept well under the influence of opium. Ice may be given to him to suck in small quantities, but he should be allowed to drink very little fluid. The treatment in fact should be the same as in a case of acute peritonitis or of ordinary intestinal obstruction.

Inflation, however, may fail, even if repeated two or three times at intervals of a few hours. When this has occurred, I am of opinion that the operation for opening the abdomen should always be performed without further delay. As far back as the year 1784 this was done successfully in Paris, in the case of a woman, aged fifty. In 1873 Mr Hutchinson advocated it in a paper read before the Royal Medical and Chirurgical Society. He had in 1871 performed the operation on a child, aged two years, who had an ileo-cæcal intussusception for a month, which was protruding from the anus. He opened the abdomen in the median line, introduced two or three fingers, and quickly draw out the invaginated mass at the wound. He then easily effected its reduction and returned it into the abdominal cavity. This was all done in two or three minutes, and the child recovered without a bad symptom.

Mr Hutchinson remarks that the cases most suitable for such an operation are those of ileo-cæcal intussusception, in which the symptoms come on slowly. In such cases there is but little tendency to sloughing and detachment of the invaginated part, so that on the one hand there is scarcely any prospect of a spontaneous cure, and on the other hand the surgeon is very unlikely to find the bowel in such a state as would render it impossible for him to proceed with the operation. But, as I have pointed out, the included portion of intestine is often highly congested, and this may seriously interfere with the complete reduction of the invagination. Dr Goodhart made an autopsy in one case, on a child, aged six months, who had died in less than twenty-four hours, and in that instance one could neither by traction nor by squeezing succeed in replacing the whole of the cæcum. The end of it still remained inverted.

Now, as I have already stated, the occurrence of hæmorrhage, with or without tenesmus, is at least suggestive of the probability that the included part of the bowel is in this condition. It therefore is a matter of importance that the disease should if possible be diagnosed before this symptom shows itself. In 1874 Dr Adcock, of Bermondsey, asked me to see a woman who had suffered for a fortnight from paroxysmal attacks of pain in the abdomen. I readily detected a tumour which presented all the characters indicative of an intussusception. She had but little sickness and no constipation. Inflation with air was at once practised; and the swelling then receded from the left iliac fossa towards the right side of the abdomen, which had been its original seat. This clenched the diagnosis, which had even before been stated in a very positive form. We therefore asked Mr Howse to see the patient. He took the same view of its nature, and after repeating the injection of air he performed the operation of abdominal section. There was no difficulty whatever in reducing the invagination. She recovered without a single bad symptom.

We now enter upon a subject—mechanical obstruction of the bowels —of which the pathological aspect is exceedingly complicated. It will therefore be convenient that we should approach it from this side. Now, of the various conditions that may occlude the gut some have their seat in its interior, others in its coats, while a third set affect it from without. And each of these will have to be considered in relation both to the small and to the large intestine.

1. The bowel may be blocked by a solid mass occupying its lumen.

In the *small intestine* this scarcely ever occurs except from the presence of a large gall-stone—one moulded upon the interior of the gall-bladder. At least six such cases are recorded in the 'Pathological Transactions;' but they must nevertheless be of infrequent occurrence, for within the last twenty years I believe that not one has occurred at Guy's Hospital. In idiots the ileum has sometimes been found obstructed by balls made up of fibrous materials that had been swallowed. Dr Langdon Down in 1866 showed to the Pathological Society a mass of interlaced cocoa-nut fibres the size of a hen's egg, which had caused death by occluding this part of the intestine; the patient, a boy aged sixteen, had had a habit of putting a shred of cocoa-nut fibre into his mouth and playing with it between his teeth. A similar case has been related to me by Dr Mackenzie Bacon. Masses of the ascaris lumbricoides, twisted and knotted together, have also been known to block up the small intestine.

It very rarely happens that ordinary articles of food, in passing along the healthy bowel, cause anything approaching to obstruction. The only instance that I know of is one related by Dr Brinton, in which an abdominal tumour, the size of a pullet's egg, was believed to be formed of a mass of half-chewed filberts. Having been first detected in the right hypochondrium, it in two days moved almost entirely downwards into the left iliac fossa and then disappeared; a few hours afterwards the bowels acted for the first time.

In the *large intestine* obstruction has occasionally been caused by concretions. Of these one variety consists chiefly of magnesia that had been taken as a medicine. A case in point was brought before the Pathological Society in 1855 by Mr Hutchinson. It was that of a lady who had the rectum blocked by a rough hard body, at least fifteen inches in circumference, which had to be broken down before it could be removed. It was made up partly of strawberry and other seeds, partly of concentric layers of what looked like a red stone, but was found to be a mixture of iron and magnesia. The patient had formerly taken sesquioxide of iron and also carbonate of magnesia in large doses, but not for twelve years before the detection of the mass in her rectum.

In persons who eat largely of oatmeal another kind of concretion is sometimes met with, consisting of a felted mass of hairs derived from the grain. Such masses have a soft velvety feel. Several specimens of them are preserved in the museum of the College of Surgeons; their nature was first suspected by Mr Clift, and afterwards demonstrated by Dr Wollaston. As might be expected, they have been found in Scotch more often than in English patients. They have not generally caused insuperable obstruction of the bowels, but have been passed after giving rise to more or less distress. In one case, that of a man named Gordon, quoted by Sir Thomas Watson, no less than thirty-two of them, varying in size from that of a hen's egg to that of a filbert, were voided at different times.

In the large intestine, again, fæcal matter sometimes accumulates to such an extent as to cause symptoms of obstruction of the bowels. A remarkable case of this kind occurred to Dr Peacock, and was recorded by him in the 'Pathological Transactions' for 1872. A man, aged twenty-eight, died in St Thomas's Hospital after an illness of six weeks, during which he had had obstinate constipation. The bowel, from the cæcum to the rectum, was found loaded with semi-solid greenish fæces, to the amount of fifteen quarts; it measured from six to eight inches in circumference. The patient had been subject to constipation from childhood, and for twelve years before his death his bowels had never acted without an enema, aperients having ceased to produce any effect. I have myself met with one case which seemed to be of this kind and which was remarkable because the patient, a woman aged twenty-two, was attacked with abdominal pain and other symptoms of obstruction of the bowels twenty-four days before her death, but at the autopsy no cause could be discovered for her illness unless it were the presence of rounded hard fæces in considerable quantity in the sigmoid flexure and rectum. This, however, was not all that had accumulated, for much had been removed during life by a large enema.

2. Obstruction may be caused by changes in the coats of the bowel itself, causing *stricture*. In the small intestine, however, one scarcely ever meets with anything that could be compared, for example, to a stricture of the urethra or even of the œsophagus. I know of one instance in which after an operation for strangulated hernia the patient suffered from continual vomiting and died in two months, and about an inch and a half of the small intestine was found by Dr Moxon to be narrowed, its coats thickened by hard white cicatricial tissue and its mucous membrane almost devoid of villi; evidently this was the part which had been in the hernial sac. I have already mentioned that Dr Moxon saw two cases in which narrowing of the small bowel was believed to have resulted from the sloughing of an intussusception, and similar instances have occurred to other observers. In the 'Pathological Transactions' for 1869 a very remarkable case is related by Dr Wickham Legg, in which the opening from the ileum into the cæcum was only just large enough to admit a No. 9 catheter, and this writer refers to some similar instances recorded by others. He supposes that in his case the stricture was congenital, which seems to me to be but little likely. Did it also result from the shedding of an intussusception? The patient, a woman aged thirty-two, had nearly all her life been liable to attacks of what was termed colic, and six years before her death she was in University College Hospital under Dr Walshe. It was then noticed that manipulation of the abdomen produced a peculiar crackling sensation, which could be both felt and heard, and which was by some compared to that

which indicates emphysema of the connective tissue, while others likened it to that which would be caused by peas shaken about in a drum. That this was due to the presence of cherry stones and other foreign bodies in the interior of the intestine was tolerably evident, for on one occasion she passed some. The same sensation was elicited by Sir William Jenner just before the fatal termination of her illness. After death the intestine was found to contain enough fruit stones to fill almost an imperial pint measure; most of them lay in the jejunum or ileum at a distance from the ileo-cæcal valve, but a few in a dilated pouch measuring seven inches in circumference, which was situated immediately above it.

Most writers state that the cicatrisation of tuberculous ulcers is a pretty frequent cause of stricture of the small intestine; and, by way of contrast, that such a condition never follows the healing of the ulcers which occur in cases of enteric fever. But I must confess that I have never seen it result from either the one or the other, and I believe that no case of the kind is recorded in the 'Pathological Transactions,' and that none has been met with at Guy's Hospital within the last twenty-five years. The nearest approach to it that I know of is a case which occurred at the hospital in 1858. A child died of phthisis after an illness of three or fourth months' duration, of which diarrhœa had been a principal symptom. There were numerous large tuberculous ulcers in the bowel, some of them extending all round it. A remarkable degree of narrowing, apparently from contraction of the peritoneal coat of the intestine, was found at several parts, and through them the blade of an enterotome could hardly be passed. In this instance, however, the symptoms of intestinal obstruction seem to have been altogether absent.

But if the small intestine is thus scarcely liable to true stricture it is very apt to be affected with another condition, which I endeavoured, in the 'Guy's Hospital Reports' ser. 3, vol. xiv, to distinguish by the name of "*contraction*," and for which Dr Bristowe has employed the designation of "compression and traction." In this the morbid appearances are of a much less striking kind than in other forms of obstruction of the bowels, and they are but little susceptible of illustration by drawings, or of preservation in pathological museums. Consequently I believe that it had remained almost unrecognised before the publication of my paper, although it is not really by any means of infrequent occurrence. The cases which I recorded affecting the small intestine were twelve in number. In four of them the affection was the consequence of chronic peritonitis, by which more or less of the small intestine was bound down to some part of the abdominal wall, or by which its coils were made to adhere among themselves. In two it resulted from the puckering caused by cancer affecting the serous covering of the bowel, and from adhesions which had formed. In one instance, a somewhat similar condition of the intestine arose as a result of tubercular peritonitis; and in these it was due to contraction associated with chronic disease of glands in the mesentery. The remaining two cases were of a less definite nature. The great peculiarity in this form of illness is that, instead of there being one particular spot beyond which the contents of the bowel cannot pass, the impediment is generally continued through a greater or less length of it. The whole of the small intestine may be matted up, so that one is unable to say that one spot more than another was the seat of the obstruction. Or there may be one or more sharp bends or twists; and it may be easy to understand how the part of the bowel above a bend, becoming distended, pressed on that below, and occluded it. Or, one portion of the intestine being fixed by adhesions, that above it may be stretched by accumulated fæcal matter, and hang down into the pelvis, so as to drag on the attached portion, and prevent anything passing through it. Two striking instances of the latter kind have occurred in Guy's Hospital since the publication of my paper. One of the former kind came under the observation of Dr Bristowe and has been recorded by him in the 'Pathological Transactions' (vol. xxi, p. 185). In it, the intestinal coils from the middle of the ileum to within a foot of the cæcum, were adherent to one another and to the brim of the pelvis by bands and filaments of false membrane, and were so entangled that their direction was traceable without difficulty, but there was no part of the bowel along which the finger could not be passed.

In the large intestine obstruction sometimes arises from its being bound down or adherent to the adjacent structures; in other words, there is an affection analogous to "contraction" of the small intestine. I related three such cases in my paper; in one each end of the loop formed by the sigmoid flexure was bound down to the spine by firm fibrous tissue; in another the impediment resulted from adhesion of the transverse colon to the neck of an umbilical hernia; and in a third its cause was that the same part of the bowel had been dragged down and fixed to the mesentery over the lumbar vertebræ.

But below the ileo-cæcal valve, an affection of this kind is exceedingly rare in comparison with *true stricture.* This last is, indeed, the commonest of all the lesions that give rise to intestinal obstruction, being present (according to observations made at Guy's Hospital) in seventeen out of fifty-one cases, or as nearly as possible in 34 per cent. Its seat is, in the majority of cases, either in the rectum or inthe sigmoid flexure; out of twenty-two cases that have occurred at Guy's Hospital, twelve have been in one or the other of these parts; while, of the remainder, three were in the descending colon, three in the splenic flexure, one in the hepatic flexure, and two in the cæcum. Thus it may almost be said that the liability of the large intestine to stricture increases regularly from its upper to its lower end. It must be borne in mind, too, that a large number of cases of disease of the rectum, of precisely the same pathological characters, are excluded from these figures because in them obstruction of the bowels was not present during life.

The affections which cause stricture of the large intestine vary greatly in their nature in different cases. Sometimes the disease consists of a simple overgrowth of connective tissue, contracting the bowel and puckering up its muscular coats. Much more frequently it is some form of malignant growth. This often has more or less distinctly a villous character. In some cases it forms a raised ring of a bright crimson colour and projecting with a smooth velvety surface into the cavity of the bowel. In other cases it is excavated by ulceration.

Histologically it may be an ordinary glandiform carcinoma, or a cylinder epithelioma. Not infrequently it is found to have undergone extensive colloid degeneration. Secondary growths in the lymph-glands or in other viscera are often absent; the reason being, without doubt (as in the cases of cancer of the stomach and of the uterus), that the death of the patient occurs at no distant period from the commencement of the disease.

3. The remaining forms of intestinal obstruction differ from those hitherto considered in the circumstance that the constricting agent has no structural connection with the coats of the affected part of the bowel, being altogether independent of them, and invested with a distinct peritoneal covering. In the small intestine the cases which come under this head are chiefly such as would be described as examples of "*internal strangulation,*" in the narrower sense of that term. These form a considerable proportion among all cases of intestinal obstruction. Thus, in Guy's Hospital, between the years 1854 and 1869, there were thirteen of them among fifty-one cases of all kinds. The exact nature of the constricting agent varies widely in different cases. In many it is a *fibrous band* of greater or less length,

attached at each end, but free in the rest of its course. This was described by Mr Gay as the "solitary band," on account of there being generally only a single one present in each case, or at most two or three of them. Such a band may pass from any one part of the mesentery or intestine to any other part, or from the mesentery or intestine to the neck of an old hernial sac, or to the uterus, or ovary, or Fallopian tube. Or it may be derived from the omentum, and pass to any of the structures that have been mentioned. Its mode of origin is somewhat difficult of comprehension, but some observations have been made which tend to show that it may be the direct result of the process by which injury of the intestine is repaired. Mr Gay records a case in which the transverse colon was punctured by a trocar in the operation of paracentesis abdominis. The patient recovered, but died many months afterwards of fever. A firm and thick band, two inches long, was then found, passing from the seat of the wound to the parietal peritoneum. And this writer quotes a case of Jobert de Lamballe's, in which a man who had been stabbed in the abdomen by a stiletto died some time afterwards from strangulation of the intestine by a band which extended from the lining of the abdominal wall to a spot in the bowel that had been injured.

But in many cases the constricting agent is not a simple band, but a cord passing from the extremity of the vermiform appendix, or of a diverticulum ilei. The latter, indeed, is the cause of fatal obstruction of the bowels in a proportion of cases which is remarkable, if we consider how seldom it is found in those who die from other causes. Among fifteen cases of internal strangulation at Guy's Hospital, five resulted from the presence of a diverticulum. This relic of the vitelline duct is always situated on the side of the bowel furthest from the mesentery, and always near the lower end of the ileum. According to Wilkinson King it is never more than from ten to twenty inches above the cæcum; but in the museum of Guy's Hospital we have a specimen which is stated to have been situated considerably higher. As Meckel long ago showed, this form of diverticulum is a relic of a fœtal structure, the omphalo-mesenteric duct, which passes in the embryo from the umbilical vesicle to the ileum. Before birth it ought to waste away entirely; but its intestinal end may remain pervious and acquire adhesion to the mesentery or to some other part. It is remarkable that this scarcely ever occurs except in males. Out of ten cases, in each of which a diverticulum was found at Guy's Hospital, only one occurred in a female patient. Strangulation by a cord attached to the end of the vermiform appendix appears to be relatively much less common. Duchaussoy states that females are more liable to it than males.

Again, a portion of the small intestine may pass through an aperture in the mesentery or in the omentum and be strangulated by it. Or it may be compressed by the pedicle of an ovarian tumour, or by the edge of the mesentery of another coil of small intestine hanging down into the pelvis.

Internal *hernia* is another cause of strangulation of the small intestine. A most remarkable and interesting form of this has been described by Treitz and by Dr. Pye-Smith under the name of retroperitoneal hernia ('Guy's Hosp. Rep.,' 1871, p. 131); in it a pouch is formed at the back of the abdomen, a little to the left of the spine, passing backwards and downwards behind the curve formed by the inferior mesenteric artery and its left colic branch. A specimen of this kind in which fatal strangulation occurred, was shown at one of the early meetings of the Pathological Society by Dr Peacock; and it is probable that another case was one in which Hilton performed an exploratory operation, and drew out a coil of intestine from an opening, apparently in the mesentery, just at the point where the jejunum became free from the spine. A different form of internal hernia was observed some years ago by Mr Cooper Forster; a knuckle of intestine became strangulated in a sac which lay close to the upper border of the obturator membrane, without passing through it.

It must also be borne in mind that in any case of what appears to be internal strangulation, the cause may be an external hernia too small to be discovered by manipulation. Hilton once opened the abdomen during life, and found an obturator hernia, the presence of which could not even then be detected in the thigh. And in a case in which I saw an exploratory operation performed, a very small knuckle of intestine was engaged in one femoral ring. I and others had previously examined the groins most carefully, but had failed to detect any hernia.

The large intestine scarcely ever becomes constricted by a band, or strangulated in any of the ways described in the foregoing paragraphs. The only cases that I have read of are two in which the sigmoid flexure was compressed by the mesentery of a coil of small intestine hanging into the pelvis, one in which the ascending colon is said to have been strangulated by a similar cause, one in which the same part of the bowel is stated to have been constricted by the vermiform appendix, and a fifth in which the cæcum was strangulated by a diverticulum.

The more loosely attached parts of the large intestine—the cæcum and the sigmoid flexure—are, however, each liable to a somewhat different form of obstruction, which is termed *volvulus.* This consists in the twisting of the loop formed by the affected part of the bowel, so that each of its ends may be said to be strangulated by the other one being wound round it. The two extremities of the loop are in fact screwed up into a narrow cord, their calibre is obstructed, and the circulation of blood in the walls of the bowel is arrested. In such cases I believe that the diseased portion of the intestine is always enormously enlarged. Thus the cæcum has more than once been found at Guy's Hospital filling nearly half the abdomen, and reaching up into the left hypochondrium. And in another instance the sigmoid flexure extended upwards in a similar way, so as to come into contact with the diaphragm, and push it and the thoracic viscera upwards. When an attempt is made to untwist the coil, it may spring back into its abnormal position with considerable force. There is some difficulty in understanding how such an affection is brought about. Both the cæcum and the sigmoid flexure are not uncommonly found floating freely and considerably enlarged in persons who have passed middle age, but who may have suffered from no marked abdominal symptoms except perhaps constipation. Probably such a condition is a necessary antecedent to the formation of a volvulus. But when it has been the cause of death, the loop is always found full of fluid and intensely inflamed. Much of the contents are doubtless the product of secretion from its mucous surface, and this must evidently have been poured out at an early period of the case and before the arrest of the circulation through the vessels of the affected part. Dr Bristowe has recently suggested that enteritis is in fact the primary condition, and that the twisting occurs secondarily. He supposes that the portion of intestine becomes first inflamed and paralysed, and that, being heavy with accumulated contents, it is then pushed aside by the pressure of the active parts around it. But this explanation seems altogether insufficient to explain the way in which the neck of the volvulus is screwed up; and it is disproved by the exact limitation of the inflammation to the part of the bowel which is twisted, since it is clear that the extent of this is itself determined by mechanical conditions.

General anatomy of obstructed bowel.—To complete the description of the pathological appearances in cases of obstruction, it must be mentioned that whereas the bowel below the seat of disease is pale, empty, and contracted, that above it is distended, often in the most extreme degree. The jejunum or ileum may become dilated until it is at least as big as the healthy colon; while the colon may reach a size which can only be described as enormous. Dr Moxon met with a case in which, after removal with its contents, the large intestine weighed nearly eight pounds; and some fæcal matter previously escaped. One effect of the distension is that in the small intestine the mucous membrane becomes forced out between the layers of the mesentery, forming rounded pouches (as in two specimens which I showed to the Pathological Society in 1875), for which I have proposed the name of "distension-diverticula." In the small intestine the dilatation of the gut diminishes more or less rapidly as one passes upwards above the seat of disease; but the large intestine may be distended pretty uniformly in its whole length; or there may be a great accumulation of fæcal matter in the cæcum, even when the obstruction is situated far below the arch of the colon, this being evidently the result of reversed peristaltic movements on the part of the bowel, by which its contents are forced backwards upon the ileo-cæcal valve.

In other respects the state of the bowel above the obstruction differs in different forms of the disease. In those in which the course is chronic, the muscular coat becomes greatly hypertrophied, forming a translucent grey layer which gradually increases in thickness towards the affected spot. In the acute forms this is wanting, but all the coats may be swollen and injected, so that the bowel feels unnaturally thick and massive. The mucous membrane is very apt to be ulcerated, especially in chronic cases; so that nearly the whole lining of the colon is sometimes found to be destroyed. Perforation is a not uncommon consequence of this; and I have known the greater part of the large intestine to be in a sloughing state, so that its contents were escaping from all parts of it. Peritonitis necessarily results from such a condition as this, unless the patient should die before there is time for it to be developed. But inflammation of the serous covering of the bowel is also very apt to occur at an early stage in the more acute forms of the disease, quite independently of rupture, and even of ulceration.

General symptoms and diagnosis.—In their clinical history, cases of intestinal obstruction divide themselves into two groups, in which the symptoms are to a great extent different, and of which the treatment has to be regulated by considerations which are distinct for each group. These are respectively *acute* and *chronic* in their course. By far the most convenient plan will be for me to take in succession all the points which concern one of these groups before I enter upon any of those which belong to the other. But first there are three symptoms common to both, which require a brief notice, namely, constipation, pain, and vomiting which after a time becomes fæcal.

Constipation may be said to be the fundamental symptom of obstruction of the bowels. It is generally absolute and immoveable, whether by purgatives or injections, so long as the disease remains unrelieved. A first enema may indeed bring away fæces which had lain in the part of the bowel below the seat of the disease, but subsequent ones almost invariably return uncoloured. In most cases, not even flatus can be passed per anum.*

Pain is perhaps always present, and it is often of extreme severity. Brinton distinguished two kinds of it, which are not met with alike in all forms of the disease. One is a constant pain, the seat of which generally corresponds more or less closely with the position of the obstructed part of the bowel, and which consequently is very often referred to the right iliac fossa. There may be some tenderness with it, but this is not usually very marked. Probably the pain itself is always traceable either to disorder of the circulation in the part of the intestine immediately affected, or to paralytic distension of that which lies above the obstruction. And, in the latter case, it may spread over the whole abdomen. The other kind of pain comes on in paroxysms. It evidently results from spasmodic contraction of some part of the bowel above the seat of disease, but, according to Brinton, only indirectly so; he thinks that its immediate cause is the increase of pressure in the distended coils of intestine close to the point of obstruction, towards which the waves of peristalsis set. If this opinion be correct, we can understand that the two forms of pain may be present in the same case and at the same part of the abdomen, even though much of the bowel may be dilated and paralysed.

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* [It must, however, be remembered that liquid fæces may pass through a stricture which nevertheless causes great distension, ulceration, or perforation of the part above it. In a case in which I had diagnosed annular stricture of the ascending colon and had arranged an operation, the passage of a large liquid motion of healthy fæces made us postpone interference. The patient afterwards died, and the cæcum was found enormously distended and perforated.—Ed.]

Vomiting occurs sooner or later in all forms of intestinal obstruction and in all cases. Its severity depends partly upon the seat and nature of the affection, but it is also liable to be greatly increased if the patient should take much liquid into his stomach. Brinton, indeed, says that in animals in which he ligatured the intestine, the quantity of fluid which they drank had more influence than anything else in determining not only the amount of sickness, but also the rapidity with which death ensued. The matters rejected in cases of obstruction of the bowels consist at first of the gastric contents, then of bilious fluid from the duodenum, and afterwards of matters derived from the small intestine down to the obstructed part, and perhaps also sometimes from the colon. These almost always constitute a thin yellow liquid, which, if the seat of disease be high in the jejunum, may throughout have merely a disgusting mawkish odour, but in all other cases the smell becomes very decidedly fæcal at last. The stench is often so powerful as to fill the room in which the patient lies, and to be almost insupportable by those about him. The cause of "stercoraceous vomiting," as it is called, has been matter of some discussion. Until a few years ago it was universally believed to result from a reversal of the peristaltic movements of the intestine, the waves of which were supposed to travel upwards instead of downwards when obstruction existed. By Brinton, however, the occurrence of antiperistalsis was denied; he showed that even though the muscular coats of the bowel should continue to contract in the ordinary way, there would be a tendency to the formation of a double current in its interior, one downwards in the outer part of its channel and another upwards in its central part, and he conceived that by this the contents of the whole alimentary canal above the seat of disease must soon be so completely mixed up that the fluid in the duodenum and even the stomach would acquire fæcal characters. Since that time, however, Engelmann has demonstrated the occurrence of reverse contractions in the intestines of animals, in which he had opened the peritoneal cavity. And I think that in all probability antiperistalsis does occur to some extent in the human subject also, under conditions of disease. Indeed, I do not know in what other way the excessive distension of the cæcum in cases of stricture of the lower part of the large intestine can be explained. It is, however, certain that the direction of the waves of muscular contraction in the bowel is not entirely reversed; for, if it were, the part immediately above the seat of obstruction would not become distended; and such substances as mercury or castor-oil, when given by the mouth shortly before the patient's death, would not be found to have passed down to the stricture.

One result of stercoraceous vomiting which I do not remember to have seen mentioned is that, during the distressing efforts which accompany it, some of the fæcal matter may be sucked into the air-passages. I have more than once noticed in the post-mortem room that this had occurred; when pressure was made towards the cut surfaces of the bronchial tubes these gave exit to little yellow cylinders which certainly must have entered them during life; and in one case of strangulated hernia, in which death had arisen from peritonitis after relief of the obstruction, the lungs contained patches of gangrenous pneumonia, which had a yellow colour in the centre due as I believed to staining by fæcal matter.

So much for the symptoms which are common to both acute and chronic cases of obstruction of the bowels. I have now to enter upon other points in regard to which these two groups of cases present wide differences. And for reasons which will ultimately appear, I prefer to deal first with the chronic.

Chronic obstruction of the bowels is in the first place characterised by the slow or imperfect development of the symptoms already mentioned. Thus the constipation is sometimes incomplete, scanty fæcal evacuations occurring from time to time. Indeed, for several weeks or even months before obstruction definitely sets in the patient often has considerable and increasing difficulty in procuring an action of the bowels; or he may have repeated attacks of partial obstruction before the one which at length completely closes the intestine. Even when the constipation is absolute it is wonderful how life is sometimes prolonged. Mr. Cooper Forster has recorded an instance in which there was not any action of the bowels for eighty-eight days. Again, vomiting is often absent in cases of this kind for some days or even weeks after the cessation of fæcal evacuations. And fixed pain may be altogether wanting, the only pain being paroxysmal.

If in such a case the abdomen be examined during one of the paroxysms of pain, it will often be found to present appearances which (as I have pointed out in my paper already referred to) I believe to be characteristic of the chronic forms of obstruction. The writhing movements of the intestine can be seen through the parietes; irregular elevations arise here and there, and are succeeded by depressions, or appear to travel from one part of the surface to another. For the production of these appearances to any marked extent I believe it to be essential that the coats of the bowel should have undergone hypertrophy; at any rate I have never observed it in cases of acute intestinal obstruction. Peristaltic movements are much more often seen in the small than in the large intestine, but I have once distinctly observed them in the latter. Even during the intervals between the paroxysms of pain, the position of the different parts of the bowel is often distinctly visible through the abdominal parietes; and it is to be particularly noted that the large intestine, when distended, does not continue to lie horizontally across the upper part of the abdomen, but bends downwards, and may form a broad loop, lying vertically and (with the dilated ascending and descending colon) filling the whole front of the abdomen. On the other hand, the coils of the ileum, under similar circumstances, are generally arranged transversely. Now, as these coils are often quite as broad as the transverse colon would be, at least under normal conditions, the uppermost one, lying horizontally just below the ribs, may be mistaken for that part of the large intestine, if the facts just mentioned be not borne in mind. I have more than once seen this error committed.

The general symptoms presented by a patient suffering from chronic obstruction of the bowels are sometimes exceedingly slight, particularly when the treatment is judiciously managed. His pulse may be quite natural; there may be no fever; he may sleep well at night. The tongue may be clean, and food may be fairly well relished.

Such a patient, however, is always on the brink of a precipice. At any moment acute symptoms may supervene which may destroy life in a day or two, or still more rapidly. Probably in many cases these symptoms depend upon the occurrence of paralytic distension in the part of the bowel above the obstruction; for I believe that this condition is sufficient to account for them. It must also be remembered that ulceration is very apt to take place in the same part of the bowel; and this is unattended with symptoms. Thus one can never say when perforation may not take place. It once happened to me to send up from my out-patient room into the medical ward a woman who had cancer of the lower part of the sigmoid flexure. She had been ill for three months. I went up to see her later in the afternoon and she appeared to be perfectly comfortable, had a quiet pulse, and presented no urgent symptoms whatever. I therefore thought that the question of colotomy might be safely deferred until the following day. In the evening the nurse was turning her over to give her an enema when she suddenly expired. About a pint of liquid fæces was found in the abdominal cavity, which had escaped from an opening in the sigmoid flexure. There were also several large sloughing patches in the peritoneal covering of the cæcum, as well as in its mucous lining.

Thus it would be difficult to fix an average duration for cases of chronic obstruction, and if one could be fixed it would be of no practical value.

Chronic obstruction is sometimes caused by impaction of the large bowel with indurated masses of fæces, but among the diseased conditions that may affect the coats of the intestines there are only two—contraction and stricture—which ever run a chronic course, and I believe that any case of which the characters are those described in the last two paragraphs must belong to either the one or the other. Now, as we have seen, contractions occur chiefly in the small, and strictures in the large, intestine. The distinction between them is therefore to be based mainly upon the points already mentioned as respectively indicating distension of one or of the other of these divisions of the intestinal canal. There is also a difference in the shape of the abdomen when distended, according as the arch of the colon is below or above the seat of obstruction. In the former case the belly is rounded, projecting well forwards, but with comparatively little fulness of the lateral and lumbar regions. In the latter case it is more broad, and if the hand be placed in the patient's loins as he lies in bed, a feeling of resistance is experienced which is wanting when the small intestine is alone distended.

In some instances a tumour can be discovered on palpation; and this, or the fact that the pain is referred definitely to one particular spot, may suggest the exact seat of the mischief. And all the signs which show that disease of the large bowel is the cause of intestinal obstruction in reality indicate more than this fact, and point to the conclusion that the affected spot is situated below, or to the left of, the hepatic flexure of the colon. So far as these signs are concerned, chronic obstruction of the cæcum or even of the ascending colon is undistinguishable clinically from that of the lower part of the small intestine; for the transverse colon does not in either case become distended. We have seen, however, that the right side of the colon is but very little liable to disease in comparison with the descending colon, sigmoid flexure, or rectum.

Now, with a view to treatment, it is of great importance that we should be able to make out whether the seat of obstruction is below the middle of the descending colon or above it. In some cases one may perhaps determine this by placing each hand in one of the patient's loins as he lies in bed, and by pressing forwards so as to "poise" the two sides of the abdomen. A fulness may be felt in the right loin which is wanting in the left; and it may be inferred that the ascending colon is distended, but not the descending colon. But I cannot say that I attach much importance to this. Another procedure which may throw light upon the question at issue is the slow injection of a large quantity of fluid into the rectum. Brinton believed that this was capable of yielding very trustworthy conclusions as to the seat of the disease. According to this writer, when a pint is the most that can be thrown up, the obstruction is at the upper part of the rectum; a pint and a half, two pints, three pints, correspond respectively with different segments of the sigmoid flexure. The descending and transverse colon can be made to receive larger but more irregular quantities. In one case, in which it was evident that a stricture occupied the upper part of the ascending colon, nine pints of fluid were always found to be the most that could be injected. But it is to be borne in mind that a stricture may be pervious to fluid injected from below, although fæcal matter may be unable to pass through it from above. Thus, in one of the cases recorded in the 'Guy's Hospital Reports' ser. iii, v. xiv, in which there was a mass of disease in the sigmoid flexure, just above the pelvis, four pints of warm water were injected, of which only a small portion returned, the rest having doubtless passed upwards through the affected part of the bowel.

It is well known that in cases of cancer of the lower part of the rectum one of the earliest symptoms is often the fact that the fæces are narrow, and the same thing is sometimes noticed when disease is situated a little higher up in the bowel. I remember one patient in whom its seat was the upper part of the sigmoid flexure, and who declared that for three months he had noticed his motions to be smaller than natural. But, whatever may be the value of this sign, even if it is constantly present when the fæces are hard, one must not forget that under conditions of temporary irritation of the colon, formed evacuations of soft consistence may be much narrower than usual without there being any permanent affection of the bowel. Moreover, there must be a point above which disease of the colon would not give rise to any change in the size of the fæces, these being moulded into shape lower down.

Digital examination of the rectum is not to be omitted in any case of chronic (nor indeed of acute) obstruction. And if there is reason to believe that the seat of mischief is the extreme lower end of the large intestine, it may be well, when the finger fails to reach the diseased spot, to adopt Mr Maunder's suggestion, and to pass the whole hand into the bowel, the patient being under the influence of chloroform. All observers appear to be agreed that introducing a long tube into the rectum is useless for purposes of diagnosis; it may catch against a fold of mucous membrane, or against the seat of obstruction, and in either case it may bend upon itself so as to appear to pass up much higher than it really does.

Besides the determination of the seat of obstruction, the pathological character of the disease is also a question for diagnosis, and one which it would in some cases be extremely important to settle, if only this were possible. We have seen that both strictures and "contractions" may be either simple or cancerous, and sometimes the discovery of a definite tumour shows that the latter is the case in a particular instance. But I doubt whether there is any other way of proving it. Cancer affecting the bowel is by no means confined to persons of advanced age. Among nineteen cases that I collected for my paper in the 'Guy's Hospital Reports,' six occurred in patients less than thirty-five years old. I have seen more than one instance of malignant disease in a person who looked well and had a florid countenance. Cancer of the bowel perhaps destroys life at too early a period of its growth to give rise to a proper cachexia. On the other hand, even if the patient's health should be broken down, one can seldom say that this may not have been the result of pain and sickness, and that the disease may not after all be of a non-malignant character.

Treatment.—In cases of chronic obstruction the patient has often been taking powerful purgatives for a considerable time before he seeks medical advice. When this is the case it may be advisable to abstain entirely from the use of such drugs, and to employ enemata only. The latter are generally of great service. In almost every instance there is at first a possibility that the disease may be merely an impaction of the contents of the intestine, and even when organic disease of the coats of the bowel is present, the systematic administration of enemata, with or without the use of gentle purgatives, not infrequently removes all the symptoms for a time. The constipation is, indeed, almost certain to return after a shorter or longer interval, and even if it should once more yield to similar treatment, the period at length arrives when the bowels remain occluded in spite of all that can be done. Then, if not before, the patient must cease to take purgative medicines, and, as a general rule, I think that it is wrong to prescribe them whenever peristaltic movements of the bowels can be felt or seen, when the abdomen is becoming at all rapidly distended, or when symptoms of collapse or of severe constitutional disturbance are developing themselves. But small doses of olive oil, as in the Mistura Olei of the Guy's Hospital Pharmacopœia,* are admissible when ordinary aperient drugs ought not to be given. Enemata may be continued somewhat longer still, but at last they too must cease to be administered.

The remedy which should now be prescribed is opium, and that not sparingly; from half a grain to a grain may be given every four hours, or even every two hours, according to circumstances, and with it a quarter of a grain of extract of belladonna, which in such cases seems to quiet, instead of exciting, the peristaltic movements.

If the patient should suffer greatly from flatulence, the application of turpentine stupes to the abdomen may now be useful. Another measure to which recourse may be had for the relief of this symptom is puncturing the intestine with a fine trocar. The late Mr Stocker long ago performed this operation, and I have seen Dr Braxton Hicks, at Guy's Hospital, let out a large quantity of flatus by this means. Moreover, the introduction of a sharp instrument into the bowel appears to act as a very powerful stimulus, for fæcal evacuations have often been passed soon afterwards, and that this is not due to the escape of the gas is evident from a remarkable case of Dr Pye-Smith's in which puncturing the abdomen in five places with a grooved needle, although neither fluid nor gas was withdrawn, led to a complete subsidence of all the symptoms of intestinal obstruction and to the prolongation of the patient's life for several months. In chronic cases there is little or no fear of the escape of the contents of the bowel into the peritoneal cavity, for the mucous membrane protrudes into the minute aperture so as to close it, but I must add that when the intestines are inflamed, I believe the procedure can no longer be regarded as innocuous.

But as soon as it is evident that a case of chronic obstruction will not yield to simple measures, the question arises whether the bowel should not be opened at some point above the obstruction, so as to establish a fæcal fistula. Now, if one can clearly make out that the seat of disease is below the descending colon, this part of the bowel should be selected for the operation. On the other hand, if there be a doubt whether the obstacle lies below the descending colon, but none that it lies below the ascending colon, the latter must of course be opened. In either case the operation of colotomy is, as a rule, successful; the peritoneum need not be wounded, and the patient's life is often prolonged for months or even for two or three years. This operation ought not, I think, to be delayed for many days after the time at which the administration of purgatives and enemata is discontinued. As a striking instance of the benefit that may

* ℞ Ol. Olivæ ʒj, Liq. Potassæ ♏iv, Aq. dist. ad ℥j. M.

result from colotomy, I may mention a case of Hilton's recorded in the 'Guy's Hospital Reports,' 1868, p. 219. There had been absolute constipation for twenty-eight days, but four days after the operation fæces began to pass through the rectum, and in a short time the wound in the loin closed. The symptoms, however, afterwards returned, and it was necessary to reopen the colon. After this a dilator was introduced twice a day with the object of keeping open the fistulous passage; but, it spite of this it again became occluded. The patient, however, who was himself a medical man, began to regain his strength and resumed his professional duties, being able to visit thirty families a day without too great fatigue. Ultimately he died of abscess in the left iliac fossa, which communicated with the interior of the hip-joint. The cause of the obstruction appeared, post mortem, to be a simple puckering of the coats of the sigmoid flexure at one spot.

It is another matter when stricture or contraction affects the small intestine or even the cæcum or ascending colon. In such cases the only thing that can be done is to open one of the distended coils in the groin, as advised by Nélaton. The peritoneum must then be opened; and the chance of establishing adhesions and forming a fistulous opening must be small, unless the serous space should have been closed by previous inflammation. This procedure has occasionally succeeded, but it can seldom, I think, be justifiable.

Acute obstruction of the bowels must now be considered. In cases of this kind the symptoms differ to a considerable extent from those which belong to the chronic forms of the disease. The constipation is always absolute; any small fæcal masses that may be brought away come from below the occluded part of the intestine. Vomiting is early and severe. Fixed pain is seldom absent. The abdomen rapidly becomes distended, but its form presents comparatively little that is distinctive of one form of obstruction rather than another. Peristaltic movements are rarely if ever to be seen through the parietes, but the form of the intestinal coils may be plainly visible.

The most striking feature of cases of acute obstruction is, however, the early development of a symptom that has not yet been mentioned; namely, *collapse*. The face becomes sunken, with pinched cheeks, and dark circles round the eyes; the extremities are covered with a cold sweat; the pulse is very rapid and small, the voice is high pitched and feeble or whispering. The patient, however, may retain perfect consciousness, and be able to lift himself up in bed until just before his death. So close may be the resemblance between the general condition of a man suffering from this form of obstruction and that which occurs in Asiatic cholera, that during one epidemic a case at Guy's Hospital was actually supposed to be one of cholera with retention of the rice-water evacuations, until on post-mortem examination the disease proved to be strangulation of the intestine.

In acute obstruction of the bowels the patient passes very little urine, and the secretion may be altogether suppressed. The late Dr G. H. Barlow, who first noticed this symptom, suggested that it might be taken as an indication that the seat of disease was situated high up in the jejunum, supposing that it depended upon diminution of the area for absorption of fluid from the alimentary canal. Subsequently Dr Habershon attributed it rather to the urgency of the vomiting which occurs when the upper part of the gut is strangulated; and Dr Brinton further pointed out that the mucous membrane above the seat of obstruction is a secretory rather than an absorbing surface under such circumstances. Both these writers admitted the fact, that when the urine is suppressed the disease is high up in the bowel. But, as was pointed out in the chapter on Cholera (vol. i, pp. 293, 295), there is reason to believe that this symptom is really one of the phenomena of collapse, and occurs in all forms of intestinal obstruction, whatever their seat, in which collapse is present.

We have now to consider in what way, and to what extent, the different conditions that may cause acute obstruction of the bowels can be distinguished from one another. And in the first place I must insist upon the fact that there is no one form of disease, capable of giving rise to obstruction, that may not present itself with acute symptoms. For instance, even in cases of stricture of the large intestine, the constipation sometimes sets in suddenly and quickly leads to vomiting and to collapse. The explanation appears to be that the bowel is then occluded, not directly by the disease of its walls, but indirectly by muscular spasm, or by the bending over of the portion of intestine above, which had gradually become overloaded with its contents. The proof of this is that, as in Hilton's case mentioned on the last page, when colotomy is performed, fæces soon begin again to pass through the natural passages. At the post-mortem examination, too, one can often pass the finger through a stricture which had caused obstinate constipation during life. As a rule, indeed, when acute symptoms are present in a case of stricture or contraction, these have been preceded by less grave symptoms for some days or even weeks. But in hospital practice it may be quite impossible to elicit this fact when the patient is admitted at an advanced stage of the disease. Thus there may be scarcely anything to distinguish the case from one of some essentially acute form of obstruction. The points of most importance as indicating that the affection is of the latter kind are its having commenced with absolute suddenness, the patient having up to that time been free from all intestinal symptoms until the moment when he was attacked.

The pathological conditions which may be regarded as the proper causes of acute obstruction of the bowels fall under four principal heads:—

1. "Constriction," or "internal strangulation," affecting chiefly the small intestine; 2, "volvulus," affecting chiefly the cæcum or sigmoid flexure; 3, "impaction of a large gall-stone," affecting the small intestine only; 4, that form of "intussusception" in which its own characteristic symptoms are absent; here the small intestine is almost always the part concerned.

Now, as regards clinical differences between these several affections I believe that very little can be said. It is to be particularly noted that in volvuli there is no delay in the occurrence of sickness, as in the other forms of obstruction of the large intestine; on the contrary, all the symptoms develop themselves with peculiar rapidity; the abdomen becomes quickly distended in the greatest possible degree, and death may occur within three or four days. Impaction of a gall-stone in the small intestine is to be distinguished, if at all, by the fact that it occurs chiefly in fat elderly women. Among the numerous causes of internal strangulation, it seems to me that no diagnosis can as yet be attempted; the only fact worthy of mention being that obstruction by a band connected with a diverticulum scarcely ever occurs except in males, and chiefly in patients under twenty years of age.

The *duration* of acute obstruction of the bowels is subject to considerable variations which (so far as their cause can be traced) appear to depend on the length of bowel which has its blood circulation arrested or is affected with paralytic distension. Mr Phillips has recorded one case in which death occurred within thirty-three hours. But I believe that such cases as this are very rare, life being almost always prolonged for three or four days, and generally beyond the first week. Indeed, one may sometimes avail oneself of this for the purposes of diagnosis; a case of obscure abdominal disease which terminates fatally within two days is much more likely to be one of perforation of the stomach or intestine than one of obstruction of the bowels.

With regard to the *ætiology* of acute obstruction there is very little to be said beyond what has been already mentioned incidentally. In the great majority of cases no directly exciting cause for the attack can be discovered, but it sometimes happens that the patient may shortly before have eaten something, as, for instance, high game or venison, which may fairly be said to have probably set up excessive peristaltic action in the bowels. In other cases a fall or blow upon the abdomen seems to have been the starting-point of the disease; it was so, for instance, in a boy who died under my care in Guy's Hospital of strangulation of the ileum by a diverticulum, and who had a bruise in the right iliac fossa, the result of his having fallen upon some large stones.

In the *treatment* of acute obstruction of the bowels the first point to be considered is whether one should recommend the performance of a surgical operation, that of opening the abdomen and searching for the band or other constricting agent, with the object of cutting it through and releasing the bowel. The analogy of herniotomy is all in favour of such a course if only one can be sure of the nature of the disease. And every pathologist has met with cases in which as soon as the intestines were exposed a band was seen, which could have been divided without the slightest difficulty. Indeed, there are a few cases in which this has actually been done during life, with the result of saving the patient's life. (See Bryant, 'Med.-Chir. Trans.,' 1867; Howse, 'Guy's Hosp. Rep.,' 1874; McCarthy, 'Med.-Chir. Trans.,' 1872; Coupland and Morris, Aug. 1877, 'Brit. Med. Assoc.')

But, before we can decide as to the advisability of such an operation, two or three questions must be answered. First, have we the means of distinguishing those forms of intestinal obstruction in which it would be of service from those in which it must necessarily fail? The answer to this may, I think, be fairly satisfactory. By carefully selecting for abdominal section cases which presented in the most typical form the symptoms of acute obstruction, one could probably make it a matter almost of certainty that the operation should not be undertaken in a case of stricture or even of contraction. By excluding cases in old females, one would reduce to a minimum the chance of finding that the cause of obstruction was a gall-stone impacted in the intestine. Volvuli and the different varieties of internal strangulation would all be fair objects for such a procedure, although no doubt some of them would be far more favourable for it than others.

But another question has now to be answered, namely, whether the cases that would thus be selected for an exploratory operation are sure, if left to themselves, to terminate fatally; or rather whether the risk to life would be greater under such circumstances than if the operation were performed. Some years ago I searched the records of post-mortem examinations at Guy's Hospital very carefully to see if I could find any case of internal strangulation of the intestine in which recovery had taken place and the patient had subsequently died of some other disease. The only instance that I could discover was that of a man who had been admitted with constipation and stercoraceous vomiting under the care of Dr Addison, and who got well and afterwards died of phthisis. But in that case, although there were two loose bridles, either of which might have strangulated the bowel, there was also adhesion of a coil of small intestine by another very short bridle, and the appendix cæci was firmly bound down. Pathologically, therefore, the case probably belonged to the class of "contractions" rather than to that of internal strangulations. Thus, so far as I know, the experience of the deadhouse lends no support to the opinion that where there is actual mechanical constriction of a part of the bowel, this is ever released by natural processes. One can, indeed, perfectly well conceive that such a thing might occur. It is evidently possible that the affected portion of intestine should be disengaged by the peristaltic movements of the portion above, or the constricting band, which is often very thin, might give way beneath the pressure to which it is subjected. When the cause of strangulation is a band attached to a diverticulum of the ileum, this is commonly in a sloughing state at the time of the patient's death; but unfortunately its tendency is to give way where it joins the bowel, so that the necessary result would be a fatal extravasation of fæces into the serous cavity.

It is, however, undeniable that recovery sometimes takes place in cases which have presented all the symptoms of internal strangulation. As an example I may cite the case of a medical student who was under my care in 1874. He was first seized with abdominal pain one Sunday morning about 7 a.m., and soon afterwards had slight vomiting. There was absolute constipation, although he took several doses of purgatives. On the Tuesday his abdomen became distended, the coils of intestine being visible through the parietes. On the Wednesday I saw him for the first time and found that his face was shrunken and that his extremities were cold. The sickness was severe; and on the Thursday afternoon he rejected a large quantity of brownish liquid which evidently came in part from the intestine; and although it did not actually possess a fæcal odour, I looked on it as indicating the near approach of stercoraceous vomiting. That night, however, he passed an offensive stool containing numerous scybala; his urine at once became copious, and all his threatening symptoms quickly passed off. As he got better, I satisfied myself that I could feel an indurated mass in the region of the cæcum, and he had a relapse of short duration, in which there was an increase of tenderness in this part of the abdomen. I have no doubt therefore that the case was really one of typhilitis and not of mechanical obstruction.

For my own part, I am inclined to believe that in all probability whenever recovery takes place after symptoms of internal strangulation of the intestine, the disease has really been not mechanical obstruction at all, but inflammation of some part of the bowel, affecting mainly its serous surface; or, in other words, a local peritonitis. I have twice seen an exploratory operation performed when the cause of the symptoms has proved to be suppurative peritonitis starting from ulceration of the appendix vermiformis. A critical review of the history of each of these cases does indeed reveal some differences between their symptoms and those which belong to true internal strangulation, but it is easy to be wise after the event. (See on this point Mr Bryant's lecture, 'Brit. Med. Journ.,' 1884, ii, p. 1182.)

I was myself at one time strongly disposed to advocate the performance of this operation, but I must confess that I am now very doubtful about it. I have seen several cases in which it has been done, and almost every one has terminated fatally. I believe, indeed, that in each of them death would have occurred within a few hours if abdominal section had not been attempted, but this only shows that if the operation is to have a chance of success it must be undertaken at an earlier period of the disease, before the patient becomes collapsed or is worn out by pain and suffering. This, in fact, is what Mr Howse recommends, and he has given several valuable directions in regard to the employment of the antiseptic method in such cases; but I fear that if such a course were adopted, the disease would sometimes be found to be typhlitis. In this case one can hardly help thinking that the operation would remove the patient's last chance of life; yet it is true that nothing but acute peritonitis was found in a patient on whom Dr Buchanan, of Glasgow, operated, and the patient began to improve from the time when his abdomen was opened, and ultimately got well. In that instance the successful issue was supposed to be due to the removal of acrid inflammatory products from the serous cavity.*

To sum up, then, I am inclined to think that but few successes appear likely to be attained by the operation of abdominal section in cases of internal strangulation, and there would be at least a corresponding number of cases which would have done well without it, and in which, instead of increasing the chance of the patient's recovery, it would rather augment his risk. The question, however, can only be settled by a wider experience.

It is particularly to be observed that whatever conclusion one may arrive at in reference to this matter must be based upon general considerations, without reference to the facts of a particular case. For upon it must depend the treatment of the disease from its commencement. If one should entertain the purpose of opening the abdomen later on, I agree with Mr Howse that the patient must not be placed under the influence of opium. This drug masks the symptoms, and prevents one's knowing what the real state of the case is; but if one should not intend to operate then there can be no doubt as to the advantage derived from the free use of opium. A dose of one grain should be given every four, three, or two hours, according to circumstances; it often affords marvellous relief. All sickness and pain may entirely pass off for the time; at the worst the patient's death is freed from the suffering which would otherwise have attended it. And if spontaneous subsidence of the disease be possible, this chance is greatly increased.

Under any circumstances not a single dose of purgative medicine should ever be prescribed, even at the very commencement of the disease, in a case presenting the characters of acute intestinal obstruction.

A course which may be regarded as intermediate between that of opening the abdomen and that of trusting entirely to the efforts of nature is the procedure of kneading the bowels, in the hope of mechanically replacing them in their proper position. This is undoubtedly sometimes followed by immediate recovery. Sir Thomas Watson relates the case of a lady who observed that the hands of two other medical men who were seeing her with him in consultation were heavy as they manipulated her abdomen; she fancied that their pressure had displaced something within, and almost directly afterwards she passed a liquid motion. Some years ago the procedure of kneading the abdominal parietes was adopted in a case which I had seen in consultation a few days before, and within a very short time the bowels acted. But striking as such cases appear, I think that in all probability recovery would have occurred even if no manipulation of the abdomen had been attempted, and it must be obvious that the treatment is fraught with danger, of which the extent cannot possibly be estimated.

Intestinal Worms.—The human alimentary canal, as is well known, affords shelter and food to several species of animals, which are commonly known as intestinal worms. By zoologists these are placed in two separate groups, Cestoidea and Nematoda, of the class Scolecida. Their differences are very great.

The Nematoda, or "Roundworms," have long cylindrical bodies; they are provided with an alimentary canal, they are males or females, and they undergo comparatively slight changes of form in the course of their development; their description will come towards the end of this chapter.

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* [The bold plan of treating suppurative peritonitis, whether dependent on obstruction, typhlitis or perforation, by abdominal section, irrigation and drainage has recently found much support. See a paper by Mr Thomas Smith in the 'St Barth. Hosp. Reports' for 1873 (vol. ix), and cases brought before the Royal Medical and Chirurgical Society, March 10, 1885, by Mr Treves and Mr Howard Marsh.—Ed.]

The Cestoidea, or "Tapeworms," are flat, riband-like creatures, made up of a number of joints, which are arranged in a line from one end to the other. They have no alimentary canal. Each joint has a double sexual apparatus, both male and female. The joints are apt to become detached, and are then capable of maintaining for a time an independent existence.

There was formerly much discussion as to the application of the term *individual* to the Cestoidea. On the one hand, a joint after its separation would seem to deserve this title; on the other hand, the animal has at one end a so-called "head," provided with suckers, and often with a circle of hard hooks which fastens itself to the intestinal wall, and which at first sight suggests that the entire tapeworm should be regarded as the "individual." And, as Leuckart admits, this view derives support from the fact that the movements of the creature take place by waves transmitted from one joint to another, large portions of it shortening or lengthening themselves at the same time, as though the joints were all under the influence of a common directing impulse. It has always seemed to me that the proper way out of the difficulty is to acknowledge that the conception of an individual, which was originally derived from a contemplation of the higher classes only, is inapplicable to a large number of the members of the lower classes of the animal world. German writers, however, have adopted a complicated mode of expression; they speak of the "head" as a "nurse;" then, by a confusion of metaphor, they call the entire tapeworm a "colony," and finally they make a point of designating its separate joints "individuals." Certain technical names are also in use; the tapeworm as a whole is called a *strobila*, the head (before budding) is a *scolex*, the separate joints are known as *proglottides*. I shall have to employ these terms presently; the reader must therefore have their exact signification impressed upon his mind.

In the course of their development the Cestoidea pass through a most extraordinary series of changes, which bring them within the scope of pathology at various points, and it will be convenient that I should give a general account of these before I pass on to describe individual species of tapeworms.

For a reason which will presently appear, the Cestoidea very rarely occur in the alimentary canal of any but carnivorous animals. Let us now suppose that a tapeworm is present in the intestine of a man, or of a dog, or cat, or other such creature. Its joints or proglottides are by no means all alike. Those nearest the head are comparatively very small, and appear almost structureless. In fact the development of new proglottides is constantly going on at this part of the strobila, and these, as they are formed, necessarily separate the scolex further and further from those which preceded them. Thus, the greater the distance from the head the older are the joints, and towards the distal end of the tapeworm they acquire a distinct sexual apparatus. This consists, as I have already stated, of both male and female organs in each proglottis, the orifices of which are generally situated side by side. There is still some doubt as to the way in which the female organ becomes impregnated; Leuckart thinks that this is generally effected by the male organ of the same joint, but others suppose that two proglottides come into contact and mutually impregnate one another. However this may be, ova are developed, and within each of them an embryo is presently formed, which is a globular body, provided at one part of its circumference with six delicate curved hooks arranged in pairs. This is enclosed in a thick shell, which in some species of tapeworm consists of several layers.

It does not appear that the ova are ever discharged from the proglottis through the "vagina;" they are, indeed, too large to be able to pass through it; they remain *in situ* until the proglottis is ruptured, so that a way is made for their escape. In the meantime this itself enters upon a more or less prolonged series of adventures. We have seen that the tapeworm is constantly forming new joints near its head. At its other end the mature joints are as constantly being cast off. Thus every proglottis in turn may be said to travel the whole length of the strobila, until by the time that its ova with their embryos are fully developed, it reaches the distal extremity and in its turn becomes detached. When this has happened, it is either discharged with the fæces of its host, or wanders out of the rectum by its own movements; or perhaps it may be ruptured while within the intestine, in which case its ova are expelled with the fæces.

Having reached the external world, the proglottides creep about for a time. If warmth and moisture befall them, they remain alive and active for some days. Leuckart supposes that they may crawl up the stalk of a plant or a blade of grass, and with this be swallowed by some herbivorous animal. Probably it more often happens that they die and become disintegrated, or that (as Dr Cobbold says) the growth of the multitude of ova within them causes them to burst. In either case the ova escape and become scattered in all directions. Some are perhaps carried into streams and ponds, others get upon the stems or leaves of plants. They retain life for several days under favourable circumstances. The immense majority of them no doubt perish, but from time to time one of them meets with the fate which is necessary for its further development.

This fate consists in its being ingested by some particular species of animal, which is generally herbivorous, but which may swallow the tapeworm ovum either in the water which it drinks or in the food which it eats. As soon as it reaches the stomach of this animal, which is in future to be its host, the ovum loses its shell, this being dissolved by the action of the gastric juice. The six-hooked embryo is thus set free, and it immediately starts upon an active migration of its own. By means of its hooks it bores through the walls of the stomach or intestine of its host. In this way it is very likely to enter some radicle of the portal vein; and, being washed away by the current of the circulation, to be carried to distant parts of the body. In other instances it perhaps continues its active movements through the tissues, until it has reached a spot far from its starting-point. However this may be, its migration ultimately ceases, and it takes up a position in some part of the body of its host, and there undergoes an entirely new phase in its development. In the first place it begins to grow and loses its six hooks, which can no longer be of service to it. It also becomes surrounded by a layer of granular matter, which is an exudation from the tissues of its host. Within four or five days from the time when a rabbit was made to swallow the ova of a tapeworm, Leuckart found, on killing it, that its liver and lungs were studded with minute white grains, exactly like miliary tubercles, but each having in its centre a tapeworm-embryo.

The embryo still goes on increasing in size, and when it has reached a diameter of 0·6 to 0·8 mm., it becomes hollow in the centre, the cavity being filled with a transparent watery fluid. From this time it presents the character of a more or less globular vesicle or bladder; and as it was recognised in this condition long before its relation to its tapeworm parent was understood, it was formerly known as a *bladder-worm*. As an example I may mention the "hydatid" or *echinococcus*, which is so often found in the human liver; and the "measles" of the flesh of pigs or sheep, the scientific name for which is *cysticercus*. All of these, I must add, are surrounded by capsules of fibrous tissue, derived from the tissues of their host, in which they lie perfectly free and unattached, but which grow as they grow, fitting tightly to their exterior and giving them the support which they need. It is from the blood-vessels of the capsule that they derive their nourishment.

After a time the growing bladder-worm begins to show a projection from one part of its inner surface, and this gradually increases in size and becomes pear shaped. Soon, four suckers make their appearance in the interior of this body, and sometimes a circle of minute hooks; it thus acquires a striking resemblance to the head of a tapeworm, and after a time a kind of neck becomes developed by which it is suspended in the interior of the bladder-worm. In strictness it should be added that the likeness is not exactly to a tapeworm head as one is accustomed to see it, but as it would appear if it were withdrawn into its body, just as the finger of a glove may be turned inside out.

In this condition the bladder-worm may remain quiescent for a lengthened period embedded in the tissues of its host. It may die there, and its remains shrivel up until only a small cheesy or calcareous relic is left. But if its host should die first, it may be set free, and then its transformations may recommence. The condition required for its further development is that it should be ingested by a carnivorous animal, with or without the tissues in which it is embedded. Thus the cysticercus or measle of pigs or of horned cattle is swallowed by men or dogs; the hydatid of sheep or men by dogs or wolves.

Having thus reached the alimentary canal of a new host, the bladder-worm enters upon a series of changes, which terminate in its conversion into a tapeworm. In the first place, the parts which have been described as resembling a tapeworm's head and neck, inverted like the finger of a glove, now turn themselves inside out. Thus, instead of being suspended in the interior of the bladder-worm, the head and neck come to project from its exterior, and may be said to have it hanging from them, so that some writers now give it the name of a "caudal vesicle." This, however, has but a very brief existence. It is speedily dissolved by the gastric juice, except a small remnant which for a time may be observed attached to the fore end of the neck. The head and neck resist the solvent action and pass on into the intestine. There they take on an active process of growth. Within a few days transverse lines show themselves on the neck and these increase in size and multiply until in the course of some weeks a jointed tapeworm or strobila is developed. The circle of changes undergone by the parasite is thus completed; we have arrived at the point from which we started at p. 214.

It must be added that in individual species some of the steps in this marvellous series of transformations deviate slightly from the account which I have just been giving. I shall mention the chief among these discrepancies in speaking of the several kinds of tapeworms. All the Cestoidea, without exception, require two different hosts for the completion of their existence. The one host in which the entozoon assumes the form of a bladder-worm, may be either herbivorous or carnivorous, but probably is most often the former. The other in which the parasite becomes a tapeworm, must always be carnivorous, since it is only when swallowed with animal tissues that the bladder-worm can enter the alimentary canal of its host.

As may well be supposed, the manner in which tapeworms are developed, and their relations to their respective bladder-worms, have only been discovered by patient investigations continued through many years. More than a century ago, in 1769, Pallas noted the close resemblance between common tapeworms and the *Cysticercus tenuicollis* from the abdomen of ruminants. But it was not until the year 1845 that the first definite suggestion as to the nature of bladder-worms was propounded. And even then the idea was not that they constituted a regular stage in the development of tapeworms, but rather that they were tapeworms which had "strayed" into a wrong animal and had consequently become dropsical and degenerated. Very soon, however, this was shown to be a mistake, and in 1851 Küchenmeister administered the *Cysticercus pisiformis* of the rabbit to dogs and succeeded in rearing in their intestines the *Tænia serrata;* he also gave the *Cysticercus fasciolaris* of the rat or mouse to cats, and found that it became developed into the *Tænia crassicollis.* And in 1853 the first experiments of the converse kind were performed by the same observer; proglottides of the *Tænia cœnurus* of the dog were given to sheep and lambs with the result that bladder-worms (cœnuri) were found in their brains, the symptoms of "staggers" being also present, which are well known to be caused by this parasite. Since that time similar investigations have been prosecuted with many different species, and the result is that we have now complete experimental proof of the relations and mode of development of many of the Cestoidea.

The number of species of tapeworm which have been known to occur in the human alimentary canal amounts to seven or eight, but of these only three or four have been met with sufficiently often to require description in a work like the present.

1. The *Tænia solium* was until recently believed to be the most common human tapeworm. When fully developed it measures from seven to ten feet in length or possibly more. Its head is of the size of a pin's head. This is provided with four suckers and with a probosis on which is a circle of about twenty-six hooks, arranged with their points outwards. They are of two sizes and are placed large and small alternately. The head is often black from the presence of pigment. The neck measures an inch in length. The joints are at first very small and they are broader than they are long. They gradually increase in breadth and still more in length, so that at about a yard from the head they are square. And towards the distal end of the strobila their length is considerably greater than their breadth. They there measure about half an inch long by a quarter of an inch in breadth. They have often been compared to melon seeds and are in fact not unlike them.* The orifices of the sexual apparatus or "genital pores" are placed in a little papilla which is easily recognised on one of the free edges, more or less regularly on the alternate sides of each successive joint. The "uterus" consists of a central passage, running in the length of the proglottis and giving off at right angles from seven to ten branches on each side which themselves give off complex dendritic branches. A simple way of observing their characters is to compress a tapeworm-joint slightly between two plates of glass and hold it up to the light. The eggs are globular and measure 0·03 mm. in diameter. They have a shell which appears to be of considerable thickness, this being, however, the result of the presence of a number of rod-shaped projections which closely cover its surface and which under the microscopic give it the aspect of being marked with a number of fine radiating lines.

The bladder-worm which forms a stage in the development of the Tænia solium is called the *Cysticercus cellulosæ* (*telæ* being understood). It is found chiefly in the pig, occasionally in the monkey, the dog, and some other animals, including man himself. In the pig it occurs principally in the connective tissue between the fasciculi of the voluntary muscles, when it is commonly called a *measle,* but also in the liver or the brain. Its relation to the tænia would be rendered probable by the absolute identity of the scolex which it contains with the head of that creature. But this point has also been conclusively demonstrated by experiments of both kinds. Van Beneden, Leuckart, and others have administered proglottides of the tapeworm to pigs; and the result has repeatedly been that the flesh of the animal has become full of cysticerci, the size of which has corresponded with the length of time that may have been allowed to pass before it was killed. Two months and a half are required for the full development of the cysticercus. From observations made by Stich it is probable that its life within the tissues (at least in man) is limited to from three to five years; he found that at the end of such a period cysticerci in the subcutaneous tissues, which had been plainly felt through the integument, became flaccid and shrank away until their presence could no longer be discovered.

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* [Hence they were called *cucurbitæ* and the worm *Tænia cucurbitina.* According to Küchenmeister, the Arabs call the complaint "Chabb-al-kar," *i. e.* pumpkin-seed.—Ed.]

The converse experiment to that of feeding pigs with the proglottides of the tænia of course consists in the administration of cysticerci to human beings. This has been occasionally done, the victims being sometimes criminals condemned to death, sometimes persons who volunteered for the purpose. Perhaps the most striking instance that I can quote is one of Küchenmeister's. He gave to a criminal twenty cysticerci on each of two occasions, one of which was four months, the other two months and a half before his execution; nineteen tapeworms were afterwards found in his intestines. A young man who of his own accord swallowed four cysticerci in Leuckart's presence began, for the first time in his life, to pass proglottides in his fæces three months and a half afterwards, and a month later took a dose of kousso, with the result that he passed two tapeworms each about two yards long.

The name Tænia solium, given to this parasite by Linnæus, was meant to imply that it occurred singly in the intestine; * and the same notion is expressed by the French title *ver solitaire.* But this is a mistake. Two or three are not uncommonly present in the same individual, and cases have been recorded in which twenty-five or even as many as forty-one have been passed by a single patient. This parasite is more common in adults than in children, and it has been noticed that it is often found in butchers and in cooks; these facts are of course just what might be expected, since it is derived from the "measle" of pork. Out of Europe it is said to have been observed only in India, Algiers, and North America. The duration of life of this tapeworm is estimated by Leuckart at from ten to twelve years. Cobbold mentions the case of a patient who was infested with this parasite for sixteen years. It has been said to have been present for as long as thirty-five years; but Leuckart thinks it probable that in such instances the worm was not the *T. solium*, but the *T. mediocanellata*, which will be presently described.

I have already mentioned that the *Cysticercus cellulosæ* is also sometimes found in the human subject, and I may add that this is the only known instance in which man is liable to both the larval and mature forms of any cestode entozoon. As a bladder-worm, the parasite is observed most commonly in the eye and in the brain, but it is very likely that it is really most frequently present in the muscles and subcutaneous tissue, where, however, it is very apt to escape notice. It appears to be found from time to time in the German dissecting-rooms. It is often solitary or present only in small numbers, but in Stich's case at Berlin more than three hundred could be felt through the skin. A person who has a tapeworm in the intestine cannot derive cysticerci directly from its ova; they must first pass through the stomach, where their shells are removed by the action of the gastric juice. Still it is remarkable that such patients do not more commonly become affected with bladder-worms than is really the case. The ova are very apt to hang about the anus and must frequently be carried thence by the finger-nails, particularly at night-time, and finally might reach the alimentary canal. Moreover, long-continued retching may bring the worm itself into the stomach. As a matter of fact, very few of those who have a tapeworm are ever known to become affected with cysticerci; but von Graefe found that among thirteen patients with cysticercus in the eye five had tapeworms.

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* [It should then have been written *T. sola*. The same notion was expressed by the specific name *T. solitaria* (Bradley). *T. cucurbitina* (Pallas) referred to the proglottides, *T. dentata* (Gmelin), *T. armata* (Brera) to the hooklets.—Ed.]

2. The *Tænia mediocanellata* has only recently been recognised as a distinct kind of tapeworm, having before been included under the T. solium. It is, however, of very frequent occurrence. The most striking distinction between the two species is in the head. This, in the T. mediocanellata, is flat at the summit and has neither proboscis nor circle of hooks. It commonly, but not always, contains much pigment deposited in rays round the suckers. It is much broader than that of the T. solium, so that it has an angular form. According to Küchenmeister its water-vascular system is more simple in its arrangement, and he gave to this species the name of "mediocanellata," believing that it had a median water-vessel in addition to the two lateral ones which other tæniæ possess; but this was apparently a peculiarity of a malformed specimen which he had examined. It is often called the "unarmed" tapeworm to distinguish it from the T. solium which is "armed" with its circle of hooks.

The strobila of this parasite also presents peculiarities which a medical man must be acquainted with, as it is often desirable that the species should be determined before the head can be obtained. It is considerably longer than the T. solium. Leuckart says that it may reach four yards in length. It is also firmer in texture and fatter, and of a darker colour towards its distal extremity. Its joints are more numerous. The sexual organs attain their full development, as in the T. solium, about the 450th joint from the head, but whereas in that species the uterus is full of ova at about the 200th joint further on, in T. mediocanellata this is not the case before the 360th or 400th joint.

The ripe proglottides are larger, measuring three quarters of an inch in length and a quarter to one third of an inch in breadth. They are more apt than those of T. solium to creep out of the patient's anus independently of defæcation. They also more generally rupture and discharge their ova while in the intestine, so that those which are passed per anum are shrivelled and empty.

But the most important peculiarity of the proglottides of T. mediocanellata is in the form of the uterus. This has from twenty-five to thirty branches on each side of its longitudinal channel (T. solium having only from seven to ten); they are necessarily packed much more closely, and they are simply forked over and over again and terminate in round, club-shaped ends, not in the notched or leaf-like broad pouches which are seen in T. solium. The eggs are not globular but slightly oval in form.

Another peculiarity of the T. mediocanellata is its liability to malformations of various kinds. Sometimes there are two or three genital pores in a single proglottis, each corresponding with a separate double sexual apparatus; sometimes the segmentation is incomplete. Sometimes a supernumerary proglottis projects by the side of that which forms part of the continuous line of joints, but the most remarkable malformation of all is one in which there are two distinct chains, united in their whole length by one edge at an acute angle, so as to constitute what may be termed a "double monster."

This tapeworm has only been recognised as a distinct species since Küchenmeister's original account of it was published in 1852. Bremser, indeed, had previously noticed that the tæniæ which he obtained from human beings in Vienna had no hooks, but he thought that they had dropped off in consequence of the age of the worms. Other observers adopted this view, although it obviously could not have accounted for the fact that all the tapeworms in a particular district should be unarmed.

When Küchenmeister took up the subject he pointed out that not only were the hooks absent, but also the proboscis which is possessed by the Tænia solium. He also drew attention to the other peculiarities that have already been mentioned.

For some time longer the source of Tænia mediocanellata remained undetermined. It had, however, been observed that the tapeworm which was known to be common in Abyssinia belonged to this species, and that the people there ate not raw pork, but raw beef and mutton. It was also noticed that infants to whom raw beef grated fine was given under medical advice got tapeworm, which at any rate in one instance was unarmed. Küchenmeister also related the case of a patient who had had the parasite ever since a particular period when he had dined several times on raw beefsteaks. Putting these facts together, Leuckart came to the conclusion that the bladder-worm corresponding with this tænia probably occurred in horned cattle. He therefore in 1861 gave part of a strobila on two occasions to a young calf. Twenty-five days after taking the first portion of tapeworm the calf unexpectedly died. All the muscles (including the heart), the lymphatic glands, and other parts were all full of minute round or oval vesicles, embedded in an opaque whitish substance which made them very much more conspicuous objects than they would otherwise have been. They looked very like tubercles, and, indeed, the affection has sometimes since been spoken of as an "acute cestode tuberculosis." The experiment has since been repeated by Leuckart and others, with the same result. It has also been shown that this form of bladder-worm has but a brief existence; if its host is allowed to remain alive it perishes and calcifies in about eight months.

The frequency of this parasite as compared with T. solium varies, as might be expected, in different countries, according as the people live more largely on beef or on pork. It is stated that in Bavaria and the south of Würtemberg the armed tapeworm is never met with, whereas in North Germany that species is said to occur almost to the exclusion of the unarmed species. In England, Dr Cobbold remarks that T. solium is more common among people of the lower class who eat much pork, whereas T. mediocanellata occurs rather in those who are better off and can procure veal or beef. But he has gradually been more and more decidedly led to the conclusion that the latter, on the whole, is really the kind of tapeworm which is most prevalent in this country.

According to Dr Cobbold another kind of human tapeworm is derived from a bladder-worm which occurs in the sheep. ('Parasites,' 1879, p. 96.)*

3. The only other species of tapeworm which is found in man often enough to require description in this work is now known by the name of *Bothriocephalus latus*. It is often called the "broad tapeworm," and formerly it was regarded as a species of tænia (*T. lata*). It is said to have been originally distinguished by Felix Plator in the seventeenth century. It is larger in every dimension than any other parasite which occurs in the human alimentary canal. It measures seventeen to twenty-six feet in length, and has from three to four thousand joints. These present the characteristic feature that their breadth exceeds their length. In the middle of the strobila they are nearly half an inch broad by one seventh of an inch in length. Towards the distal end they increase in length and diminish in breadth until at last their form is almost square. This tapeworm is peculiarly transparent looking. It has a longitudinal projecting ridge traversing its whole length. Its head is unarmed; it is club-shaped, and has two

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* [He has named it *Tænia tenella*, a name previously applied by Pruner, but to a "bad species." Another species, *T. nana*, was once discovered by Bilharz in large numbers in the intestine of a boy at Cairo. *T. ecchinococcus* has never been found as the strobila in man.—Ed.]

deeply-grooved longitudinal suckers, one on each side. The reproductive organs differ altogether in appearance from those of the tæniæ. The genital pore lies in the middle of each segment, opening upon its ventral surface. The uterus is an unbranched tube, which is bent on itself four or five times each way. When distended with ova its loops are flattened against one another, so that it resembles a rosette. Its ova are larger than those of the tæniæ; they measure 0·07 mm. in length, and are oval in form, with an operculum or lid at one end which rises to allow the escape of the embryo.

A peculiarity of this tapeworm is that its joints do not come away singly, but that portions of the strobila from two to four feet in length are expelled with the fæces.

The Bothriocephalus latus is almost, if not quite, limited to the inhabitants of certain countries of Europe. The locality for it which is best known is the western part of Switzerland; in Geneva one person in every four is said to harbour it in his intestine. It also occurs in the north-west of Russia, in East Prussia, in Sweden (in one province of which the whole population is said to be infested with it without exception), in Poland, Holland, and Belgium. Leuckart speaks of its having been observed in persons living in London, and implies that this has sometimes been the case even in those who had not been in any country whence they could have obtained the parasite; but I believe that this is very doubtful. It is not impossible that the Tænia mediocanellata may have formerly been sometimes mistaken for it. Leuckart remarks that muscular contraction may shorten the joints of that species, and anyone accustomed to the comparatively small T. solium might assume that a worm so much larger was the so-called T. lata.

The source from which this parasite enters the human body has not yet been certainly determined. The observation has long been made that the districts in which it is met with are low-lying regions, situated either near the sea, or at least near some large lake or river, and it has been suspected that the corresponding bladder-worm inhabits either some kind of fish, or possibly a fresh-water mollusc. Salmon, trout, and bleak have especially been mentioned as likely to prove to be the resting-place of the immature form of the bothriocephalus. Such a view derives some support from the fact, first discovered by Schubart, but more fully made known by Knoch in 1862, that by keeping the ova several months in water each of them gives out an embryo possessing the usual six hooks, but enclosed in a membrane which is completely covered with beautiful long delicate cilia. These enable it to keep up a constant rotatory movement, like that of a volvox. After four to six days, it escapes from the ciliated membrane and becomes free. Its further fate has as yet eluded observation. Knoch, indeed, thought that he had proved that the administration of proglottides of bothriocephalus to puppies led to the direct development of the tapeworm in their intestine, but the validity of his experiments is disputed by Leuckart.*

Symptoms of tapeworms.—The effects of the presence of a cestoid worm in the human intestine are of a somewhat vague kind. Persons in robust health, and children, generally experience no discomfort whatever; it is only when proglottides or portions of their strobila are evacuated that a suspicion arises that they are otherwise than perfectly well. And, as Leuckart remarks, when this has once happened, the patient often begins for the first time to complain of pains and other symptoms, of which nothing had before been heard, although the parasite must have been present for several months. The sensations which are said to have been directly caused by it are described as an "uncomfortable feeling in the abdomen," "a colicky pain," a "gnaw-

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* [Another species, *B. cordatus*, has been more than once observed in human beings in Greenland, and *B. cristatus* twice in France.—Ed.]

ing pain at the epigastrium, especially when the stomach is empty or after certain kinds of food." Sometimes the patient is convinced that he can feel the movements of the worm; and, in reference to this, it is to be said that the sluggish contractions of the creature outside the body give no idea of its activity while under the influence of the warmth of the interior of the intestine. Leuckart particularly speaks of the strong peristaltic motions of its segmented body, of the continually varying play of its suckers, and of the bendings of its neck. It always hangs downwards towards the lower part of the intestine, but sometimes it is bent on itself, or even rolled up. Leuckart mentions that in the dog he has sometimes observed injection of the mucous membrane, separation of the epithelium and even ulceration, produced by tapeworms; but I should rather doubt whether such changes in the human intestine, even if they occur, would go further than the mere presence of the parasite towards explaining any symptoms that might be observed. Foulness of the breath, an irregular or craving appetite, constipation, or, very rarely, diarrhœa, are also said to be caused by the existence of a tapeworm in the human subject.

Morbid sensations are also sometimes produced at distant parts,—itching of the anus, itching of the nose, so that the patient is always picking it, headache, giddiness, lassitude, a tendency to faintness. As might be expected, such symptoms are observed chiefly in persons of nervous irritable temperament. Grinding of the teeth at night is another symptom, and patients have been known to have hysterical fits, epileptic fits, and even maniacal attacks, which have been cured by the expulsion of the worm. Dr Graves relates the case of a young lady, who was attacked with what were regarded as alarming symptoms of bronchitis. She had a dry, hollow, cough, which was repeated every five or six seconds, night and day, whether she was asleep or awake. Bleeding, tartar emetic, blisters, antispasmodics, were tried in turn, but without result. Dr Graves and her medical attendant gave up the case in despair. At last she had a sudden attack of colic, for which an old servant of the family gave her a full dose of oil of turpentine with castor-oil. She passed a large mass of tapeworm, and from that moment every symptom of pulmonary irritation disappeared. One is not justified, however, in prescribing anthelmintics indiscriminately in all cases of spasmodic nervous affections for which one happens to be unable to find a sufficient cause, but one should at least not forget to inquire as to the presence of worms in such cases. The bothriocephalus is said to give rise to more marked symptoms than the tæniæ, but even it may be altogether latent. Bremser mentions the case of a Swiss, who had been eleven years away from his native country before he discovered that he was the bearer of this parasite.

Prophylaxis.—To prevent the development of tapeworms in the human intestine two distinct measures may be taken, which, however, scarcely apply to the bothriocephalus, since the seat of the corresponding bladder-worm has as yet only been guessed at. In the first place, meat which is observed to contain cysticerci should not be eaten at all; and in the second place, all meat should be subjected to such processes, before being eaten, as will destroy any cysticerci that may chance to be present. Measly pork may often be easily recognised; the bladders are of considerable size and may be present in very large numbers. But it is remarkable that in the flesh of horned cattle cysticerci have never yet been seen, except after the experimental administration of proglottides of T. mediocanellata to the animals. The reason evidently is that an ox or heifer is much more particular in what it eats than a pig, and consequently that its only chance of being infected with the cysticercus is by swallowing stray ova on the leaves of the plants which it eats or in the water which it drinks.

The second precaution against tapeworms consists in eating only meat which is thoroughly well cooked. The cysticercus cannot survive the temperature of boiling water. For more reasons than one people should take especial care not to eat sausages which are underdone in the middle. Pork or ham which has been thoroughly smoked or salted may, it is said, be safely eaten, even though it may not have been cooked.

Several physicians in this country have recorded instances in which persons have been infected with a tapeworm who have been addicted to eating meat raw. But the most striking instance is that given by Kaschin, of the Bürater of the Baikal. These people live almost exclusively upon flesh, which they neither properly clean nor thoroughly cook, and which they eat from tables that are never washed, and that are used also for cutting up the meat. Even when stationed as Cossacks at Irkutsk, so that many of them had been away from their native country for years, they were infested with tapeworms to such an extent that in 130 autopsies only two bodies were found to be free from the presence of the parasite; often there were several, and once as many as fifteen, in the intestines of the same individual.

The *curative treatment* of tapeworm consists in the administration of some substance which has the power of killing the creature without injuring the intestine. And at the present time, no substance is used so largely for this purpose as the liquid extract (or "oil") of male fern. Its dose is generally said to be from fifteen to thirty minims, but at Guy's Hospital we have been in the habit of giving a drachm or a drachm and a half. Dr Gull, many years ago, published in the 'Guy's Hospital Reports,' a series of 200 cases treated with this drug with much success. I have never seen it do any harm, but Dr Cobbold speaks of it as causing irregular and violent effects on the nervous system if its dose is at all large. Another useful drug, derived from Abyssinia, is kousso, which consists of the dried flowers of the *Brayera anthelmintica;* from a quarter to half an ounce of this is infused in boiling water, and it is swallowed, powder and all. Oil of turpentine, again, is often effectual, of which from half an ounce to two ounces may be taken for this purpose; a single large dose is less apt than repeated small doses to cause the strangury which is well known sometimes to result from its administration. A decoction of the bark of the pomegranate root is another valuable anthelmintic; the direction is that three or four doses of from one to two ounces each should be given at intervals of about half an hour; it often causes faintness and giddiness.

Whatever medicine may be chosen, I think it must be advisable that the patient should fast for several hours before taking it. But Dr Cobbold objects to this. The intention is that the alimentary canal being empty the drug should with more certainty come into contact with the tapeworm. And for the same reason a dose of castor-oil is sometimes given three or four hours before the anthelmintic.

The administration of either of the remedies that I have mentioned almost always brings away a large portion of the tapeworm, if there be a fully developed tapeworm in the patient's intestine. But, unfortunately, the strobila very commonly breaks at the neck. The head then remains behind; and as it still retains its vitality, it at once begins to form fresh segments. Now, if the parasite belong to either species of tænia, it is remarkable that after such an accident, an interval of three months (Dr Cobbold says thirteen weeks) is almost invariably found to elapse before proglottides again begin to be passed. I have repeatedly known this to occur on almost the very day which had been predicted for it. This period of three months corresponds exactly with the length of time which is required for the full development of the tapeworm from a cysticercus; it therefore follows not only that under the influence of anthelmintics the line of fracture is constantly at one part of the worm, but that it is quite close to the head. It no doubt sometimes happens that the creature breaks in the middle, particularly if the dose of the anthelmintic is inadequate. But this, at any rate, may be said,—that if a portion of tapeworm be brought away, in which part of the narrow neck is recognised, and if the patient should in much less than three months begin again to pass proglottides per anum, it is certain that more than one tænia is present.

The patient must always be told to look very carefully in his evacuations for the head, the appearance of which should be described to him. An enthusiastic practitioner may himself search for it. Dr Cobbold recommends that the whole mass of fæces should be passed through a sieve. If the head be not discovered, the patient may either wait for three months to learn whether the treatment has been effectual, or he may take a second dose. I should have thought it doubtful whether drugs would act satisfactorily upon a tapeworm of which nothing but the head is left; and it would be obviously very difficult to obtain evidence as to the frequency with which one might effect a cure under such circumstances. But Dr Cobbold relates one instance in which, having brought away almost the whole of a tapeworm with one dose of extract of male fern, he gave another dose the next day and actually succeeded in finding the head with its four suckers in the fæcal mass passed by the patient.

The remaining species of intestinal worms belong to the Nematoda.

The *Ascaris lumbricoides*, as its name implies, is somewhat like the common earth-worm (*lumbricus*). When alive it is of a reddish-brown colour with a tinge of yellow, but after its death this colour slightly fades, and it becomes greyish. It has a disagreeable smell, which cannot be removed by washing, and which, according to Leuckart, is due to an odorous principle having its seat in the deeper muscular layers of the body.* The female is fifteen inches long; the male, which is comparatively seldom met with, measures only ten inches; its circumference is also much less than that of the female. They are cylindrical in form, tapering at each end, but rather more gradually towards the head than the tail.

The life-history of this parasite has not yet been completely ascertained. The female discharges ova which certainly do not undergo any development while they are in the human body. After their escape with the fæces, however, an embryo slowly appears in them if they are kept in water or in moist earth. Davaine and others formerly supposed that the ova were swallowed in this condition either in drinking water or upon uncooked vegetables or fruit, and that their shells having been removed in the stomach the embryos gradually became developed into full-grown worms. But experiments made for the purpose of confirming this hypothesis have uniformly failed. Several German investigators have deliberately swallowed large numbers of ova and have given them to children, but no specimen of the ascaris has hitherto been obtained in this way. Another possibility is that the embryos escape from the ova and enjoy an independent existence for a time before they enter the human body. Thus Dr Paterson, of Leith, is quoted by Dr Aitken as having observed that certain families who drank the water of a particular well were very subject to the parasite, whereas towards the other end of the same street families who drank the pure water supplied to the town of Edinburgh were free from it. The well-water came from a dirty pond in the vicinity, and in it numerous minute vermiform animalculæ existed, which perhaps were larval ascarides. But Leuckart lays stress on the fact that the embryos of Ascaris lumbricoides show little or no tendency to escape from the ova, and that their organisation is not like that of embryos which are destined to maintain an independent existence. He therefore thinks it most probable that the ova are swallowed by some intermediate host—perhaps a worm or the larva of some insect—and that within the body of this animal the embryos pass through such further changes as may prepare them to undergo their complete development on being afterwards transferred to the human digestive canal.

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* [Dr Bastian and some others have suffered from irritation of the eyes, sneezing, and other symptoms like those of hay-catarrh from dissecting this worm.—Ed.]

The Ascaris lumbricoides appears to infest the human intestine in all parts of the world, but certain populations and classes of people are much more liable to it than others. It is rare in infants under a year old, although Leuckart refers to one instance in which it occurred in a child of eleven weeks. Children between three and ten years of age afford the most numerous examples of it. It is more common in rural districts than in towns, and particularly in low and damp localities. It is met with more often in the autumn than at any other season; this Leuckart connects with the hypothesis that eating of summer fruits is in some way concerned in introducing it into the human body. Persons who are poor and dirty are more subject to it than those in better circumstances. In the insane it is very often found. Vix found that among thirty lunatics of dirty habits in the Hofheim Asylum there was not one who was free from this parasite. In the Southern States of North America, the West Indian islands, Cayenne, and Brazil, the negroes at all ages are with scarcely an exception infested with Ascaris lumbricoides. It is also much more common in some parts of Europe than in others, particularly so in Finland and Holland.

The Ascaris lumbricoides inhabits the small intestine. It may be solitary, or there may be two, three, or any number of them. When numerous they often cohere together in packets, and they have sometimes been found filling almost the whole of the intestines. Children have been known to pass some hundreds of them in the course of a few weeks. Cruveilhier found more than a thousand in the intestine of an idiot.

It is considered probable that each individual worm remains only a few months within the body of its host. If they pass down into the large intestine they are voided from the anus, either alone or with the fæces. If they make their way upwards into the stomach they are generally vomited. Sometimes, however, one is discharged through the nose; and it has even been known to enter the larynx, and cause death by suffocation. A curious point, to which Dr Cobbold has especially drawn attention, is that the Ascaris lumbricoides is very apt to insinuate itself into any kind of small ring that may be swallowed by its host, such as the eye of a lady's dress or the shank of a button. A single worm has been found with two buttons attached to it. This peculiarity goes far towards explaining the fact that the parasite sometimes makes its way into the bile-duct or gall-bladder, setting up jaundice or even suppuration in the liver. In other cases it has escaped into the peritoneal cavity through the floor of an intestinal ulcer, supposed to have arisen independently. And in yet others it has been found within the cavity of an abscess generally situated either at the umbilicus or in the groin. There has been much discussion whether or not the inflammation has in such instances been originally set up by the presence of the worm; a strong point in favour of this view is that after its escape the abscess has generally been found to heal, thus terminating much more favourably than an ordinary fistulous opening from the bowel would be likely to do.

The *symptoms* produced by these worms vary according to the number of them which are present and the irritability of their host. It is only in a very delicate subject that a single Ascaris lumbricoides, or even two or three, would cause any appreciable discomfort. When symptoms do arise they are generally such as indicate irritation of the intestinal mucous membrane, pain in the abdomen (especially in the umbilical region), nausea, foulness of breath, irregularity of appetite, tumidity of the abdomen, and the presence of mucus in the stools. It is true that in the bodies of those who have died from other causes and in whom worms are found the intestine does not generally present any morbid appearances that can be attributed to their presence; but Barthez and Rilliet say that they have seen the mucous membrane reddened by vascular injection at points occupied by several round-worms, and not anywhere else.

More serious cases have been recorded in which masses of the parasite, rolled up together have caused fatal ileus. In a different class of cases, the Ascaris lumbricoides has given rise to reflex symptoms similar to those which have been described as effects of the presence of tapeworms: dilatation of the pupils, swelling of the eyelids, squinting, irritation of the nostrils, grinding of the teeth during sleep, &c.

In clinical *diagnosis*, there are two sets of conditions under which one has to deal with this parasite:—1, where a patient, generally a child, presents some of the symptoms just enumerated, which, in the absence of any more obvious cause, are supposed to be possibly due to its presence; and, 2, where one ascaris is said to have been vomited or passed per anum, and there is a question whether there are still others in the intestine. In the second class of cases one has sometimes to bear in mind that impostors have been known to bring earth-worms with them which they pretend to have passed from the bowels. The true lumbricus, however, is readily distinguished from an ascaris; it is much redder, it tapers less at its extremities, and it has rows of small bristles which aid it in locomotion; its mouth is a short fissure on the under surface of its rounded head, whereas the mouth of the Ascaris lumbricoides is a triangular aperture at one end of the animal, surrounded by three tubercles or lips.

When the presence of this parasite in the intestines is suspected, from whatever cause, a common practice has been to give a dose of medicine on the chance that it may bring away an ascaris. But Leuckart and others have pointed out that the question may very readily be answered by a microscopical examination of the patient's fæces, which, if the worm is there, are sure to be full of its ova. It has been calculated by Eschricht that in the female ascaris there are at one time about sixty millions of eggs, and if these were a year in being discharged from its genital passages, the patient's evacuations would contain 160,000 of them every day. No wonder, therefore, that a single microscopical specimen often places the presence of the parasite beyond dispute. The ova are elliptical in form, measuring $\frac{1}{340}$th of an inch by $\frac{1}{440}$th of an inch; and they are of a dirty brownish colour and nodulated on the surface, from the presence of a thick layer of an albuminous substance deposited outside their proper shell. In illustration of the value of this method of diagnosis, Dr Ransom gives (in his article on the entozoa in 'Reynolds' System of Medicine,' vol. iii, p. 197) the case of a child who was admitted into hospital for abdominal pains and disordered digestion, and because she had passed two round-worms previously. The evacuations contained the eggs of the parasite. Medicines on several occasions brought away one or more specimens of the ascaris, and her symptoms entirely disappeared. But ova were still detected in the stools, and therefore she was kept under treatment three months and a half longer, until seventeen worms in all had been passed. No more of the ova could then be discovered and she was accordingly sent out of the hospital.

In *treatment*, the medicine which appears to possess more power than any other in effecting the expulsion of the Ascaris lumbricoides is santonine. Its dose, for an adult, is from three to six grains twice daily, and for a child one.

to three grains. While it is being taken an occasional purgative should also be prescribed. An occasional inconvenience produced by santonine is a curious temporary disturbance of vision, objects appearing of a yellow, green, or blue colour. The urine may also be reddened, but that is of no consequence. Dr Cobbold says that it sometimes produces tenesmus, spasms, and even hæmorrhage from the bowels, so that it should not be too long continued. Dr Ransom speaks of *Dolichos pruriens* and oil of turpentine as being also worthy of trial. As to preventing this parasite from entering the body, all that can yet be said is that one should be careful to drink only pure water and to have all solid food thoroughly cooked.

The allied species *Ascaris mystax* has occasionally been observed in the human intestine both by German and British helminthologists.

The *Oxyuris vermicularis*, or "thread-worm," is very much smaller than the entozoa of which I have hitherto been speaking. It was formerly called Ascaris vermicularis, and in England this name is not yet altogether obsolete, for thread-worms are still commonly called "ascarides." There is not much risk of confusion in this use of the term, for medical men never have occasion to speak of the A. lumbricoides as present in large numbers in a living patient, but it is incorrect according to modern zoological classification, and it is liable to mislead the student. Etymologically one might indeed plead that the word (from ἀσκαρίζειν, to leap) is applicable rather to the thread-worm, which performs brisk movements, than to the comparatively sluggish round-worm.

The oxyuris may best be compared to a small piece of fine white thread. The female measures four tenths of an inch in length, the male one sixth of an inch. The latter is much less often seen and was formerly supposed to be very rare, but now it is supposed that there is about one male to every nine females. They taper towards the tail.

Thread-worms occur only in the large intestine; they are often present in vast numbers. They derive their nourishment from the fæcal matter of their host; the yellow colour can plainly be recognised in the interior of their bodies. They may lie singly in the mucus lining the interior of the bowel, or they may be matted together with this mucus into little balls. The ova of this parasite are oval in form, flattened on one side, with a smooth surface. They measure $\frac{1}{1100}$th of an inch by $\frac{1}{490}$th of an inch. Unlike those of the Ascaris lumbricoides, they have embryos in their interior at the time of their liberation from the parent worm. It might therefore be supposed that no impediment existed to the multiplication of the oxyuris within the human intestine for an indefinite period. But Leuckart and other modern observers believe that the ova are incapable of undergoing development until they have passed into the external world and been swallowed by the same or by another individual. One is at first startled when one is told that every single thread-worm that a child's intestine may contain represents an egg which the child must have taken into its mouth. But, as Leuckart points out, this theory is supported by the analogy of all other parasitic worms, none of which are capable of reproducing themselves indefinitely *in situ*. Indeed, these creatures produce such immense quantities of ova that there would be no limit to their numbers were not their development subject to some such conditions as are suggested by the theory in question. Leuckart further observes that one never finds *young* oxyurides in numbers bearing any proportion to those of the ova, whereas, if they arose directly out of them, they ought to be present in comparatively large numbers. He tested the point, as far as he could, by swallowing a few ova and giving some to those of his pupils who volunteered to share in the experiment. At the end of the second week three out of the four individuals experimented on began to pass thread-worms.

This question is one of considerable importance in reference to the measures which should be adopted for preventing the occurrence of oxyuris. This parasite creeps out of the rectum of its host, especially at night; it wanders among the folds of the anus, and in women often passes into the vagina. It is therefore quite conceivable that it might pass or be carried to the anus of another individual sleeping with the one from whence it came. Küchenmeister supposed that this was the way in which the worm obtained access to the human body; he thought that a single female oxyuris passing from one bedfellow to another might make the latter the victim of this parasite for the rest of his days. The more modern school of helminthologists also attach importance to the emigration of thread-worms from the rectum. It is noted that they and their ova often become adherent to the skin and hairs in the neighbourhood of the anus; they dry up, and ultimately break down into a fine dust, containing enormous numbers of ova still capable of springing into life if brought under suitable conditions. Thus every opportunity is afforded for what may be termed "self-reinfection." Again, every fæcal evacuation of a person infested with thread-worms probably contains hundreds of thousands of their ova. They must be carried into drinking-water, taken up by flies, deposited upon vegetables and fruit, and so in a thousand ways gain access to the human alimentary canal.

The *symptoms* produced by thread-worms are not like those caused by other entozoa, being chiefly the effects of the irritation which they produce by creeping about the anus and genital organs of their host. Chief among them is a sense of heat and tingling or itching at the fundament. This comes on at a particular time, generally about 9 or 10 p.m., when the patient is in bed, but sometimes before he has retired to rest. Marchand quotes the account which a man gave of his own sufferings, as follows:—"Every evening about 5 or 6 o'clock, when I first feel the worms, I become pale and troubled, and sometimes I have even shivered; my companions often notice it; I am restless and obliged to walk about; even if I am at a place of entertainment I leave instantly and hasten to employ a cold enema; this does not always give me relief and I am in torture; I tear my perinæum and scrotum. I am obliged to micturate every instant." Irritability of bladder is indeed well known to be sometimes caused by the presence of thread-worms, and there is reason to believe that they sometimes excite sensations of a sexual nature, priapism, and nymphomania, and that they may indirectly lead to the practice of masturbation. Another symptom may be tenesmus; and the fæces may contain a large excess of mucus. Cruveilhier recorded the case of a child who was awakened every night at the same hour by an agonising pain in the anal region, so that he screamed and writhed about in bed. The periodicity of the attacks led to the administration of quinine, but with no success, until the part was looked at, when the cause was at once discovered.

To rid a patient of thread-worms is a less easy matter than might be expected; and there is still some uncertainty as to the kind of *treatment* that is most successful. Until lately it has been taught that the rectum and sigmoid flexure were the parts of the intestine chiefly infested by this parasite; it was known that it might be found as high as the cæcum, but this was regarded as exceptional. The older writers, therefore, recommended enemata for their removal. Sir Thomas Watson remarks that it would be a very roundabout course to introduce at one end of a twisted tube several yards long, substances which are intended to act upon animals which live quite at its other end. He says that he has often relieved patients of them by prescribing simply the infusion of quassia as an injection. Lime-water, solutions of chloride of sodium, of perchloride of iron, and many other substances have been recommended by others for the same purpose. The rule has generally been that an enema should be given every third or fourth day for two or three weeks. Of late, however, the opinion has been gaining ground that the oxyuris occurs in the cæcum and in the upper part of the colon more generally than had been supposed; Dr Cobbold even says that the cæcum is its headquarters, although I do not know what proof there is of this. He therefore now recommends active saline cathartics, repeated for several days in succession, and large draughts of infusion of gentian, &c. He mentions as a useful practical hint that the introduction of a little mercurial ointment within the verge of the anus as the patient retires to rest will effectually prevent thread-worms from creeping out of the rectum, but this plan must require some caution lest salivation should follow. Sir Thomas Watson speaks of affording relief to the itching by the application of oil or of a towel wetted with cold water to the fundament. Children, particularly about five or six years old, are much more commonly infested with thread-worms than adults, but the former are often very easily freed from their presence, whereas in the latter, when this parasite does occur in sufficient numbers to be troublesome, it is apt to resist remedial measures with great obstinacy. If the modern view of the life-history of the oxyuris be correct, it is of infinite importance that the most scrupulous cleanliness should be maintained in all the surroundings of a person infested with it. But we cannot at present hope for such success in this direction as would justify Dr Ransom's remark that a person who adopted the requisite precautions against reinfection from himself or others would probably get well in a few weeks without treatment of drugs.

Another nematode worm, the *Trichocephalus dispar*, has its seat in the cæcum. It is remarkable for its very long and thread-like neck, which forms about two thirds of its whole length of one and a half to two inches. This parasite appears to give rise to no symptoms; and it has scarcely ever been discovered in the evacuations. It therefore possesses no clinical interest. But as its ova may be found in the fæces, I may mention that they are bluntly spindle-shaped, with transparent ends, and that they measure 0·023 mm. in breadth by 0·051 mm. in length.

Of infinitely greater importance, so far as practical medicine is concerned, is the *Sclerostomum duodenale*;* except, indeed, for the circumstance that it is not found in this country. It is occasionally met with in Italy, where it was discovered in 1838 at Milan by Dubini; and occurs very commonly in Egypt and in Brazil. At a meeting of the Pathological Society in 1867, some specimens of it from the latter country were exhibited by Dr Hermann Weber. (See his paper, with figures, 'Path. Trans.,' xviii, p. 274.)

In Cairo, Bilharz found it in almost every dead body which he examined. It inhabits chiefly the jejunum, generally lying between the valvulæ conniventes, with its mouth firmly fixed in the mucous membrane by means of four teeth which it possesses. It is also sometimes found rolled up in the interior of small cavities which separate the mucous from the muscular coat of the bowel. The female occurs in larger numbers than the male; the former measures seven tenths of an inch in length, the latter four tenths of an inch.

The sclerostomum lives on blood, which it sucks from the intestinal vessels. The contents of its alimentary canal are consequently of a bright red colour, which is visible through its transparent skin. As hundreds and even thousands of these parasites are sometimes present in the same individual, it is not surprising that they should give rise to a profound anæmia. Indeed, the cavity of the small intestine contains a quantity of extravasated blood, which has perhaps escaped from punctures made by worms which have afterwards moved to other spots.

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* Also known as *Ancylostomum duodenale* (Dubini), as *Strongylus* or *Dochmius duodenalis* (Leuckart, Diesing), and as *Str. quadridentatus* (von Siebold).

It was Griesinger who showed, in 1854, that the form of anæmia known as Egyptian chlorosis was due to the presence of this parasite. ('Arch. f. phys. Heilkunde,' xiii, 557.) Wucherer, of Bahia, has since shown, in 1866, that the parasite gives rise to a similar complaint in Brazil and also in the Comoro Islands.*

The symptoms of this "tropical anæmia" appear to be identical with those of other forms of anæmia. They may last for years, until the patient is at length carried off by diarrhœa or pneumonia, or some other accidental malady. Much more rarely the chlorosis proves directly fatal after causing dropsy. The medicines which have been suggested for the treatment of this disease are chiefly oil of turpentine, assafœtida, aloes, and iron, but Dr Weber says that none of them have proved to be permanently successful. If the patient can be removed to another climate and placed under favourable conditions, recovery may take place. One would have expected that the disease should sometimes have been seen in England in persons recently arrived from Egypt, just as a somewhat analogous affection, due to the Bilharzia, is often brought to this country from South Africa. The Sclerostomum was the cause of severe epidemic anæmia among the workmen in the St Gotthard Tunnel in 1880 (see a paper by Dr Bugnion, 'Brit. Med. Journ.,' March, 1881, p. 882). An allied species produces aneurysms in blood-vessels after it has wandered from the intestine, in horses, asses, &c.

I must next pass on to speak of the *Trichina spiralis*, a nematode which is truly an intestinal worm, although until recently it was only known as being now and then found unexpectedly in the muscles of the human body. How it found its way into the muscles was for a long time a puzzle which exercised the minds of the ablest zoologists. But, as we shall presently see, its life-history is not really so very unlike that of the other nematodes which infest the human intestine. The principal difference is that, instead of its ova being discharged per anum, they develop within the parent worm into young trichinæ, which as soon as they are born begin an active migration among the tissues of their host.

Tiedemann, in the year 1822, was the earliest observer who took note of the presence of a number of white stony concretions in the human muscles, but he did not describe them further, and Leuckart doubts whether these were trichina capsules. In this country Hilton was the first, in 1832, to record the fact that he had met with them.† He could not determine their precise nature, but thought that they were probably very small cysticerci. Next Mr James Paget, then a student of St Bartholomew's, independently described them. Two years later Owen showed that they were hollow capsules, and that each of them had coiled up within it a minute nematoid worm, to which he gave the name of *Trichina spiralis* and fully described its anatomy.

The capsules themselves are just visible to the naked eye; they measure $\frac{1}{78}$th inch in length and $\frac{1}{130}$th inch in breadth. They are perhaps best described as lemon shaped. Their long diameter is always in a line with the muscular fibres among which they lie. They occur in all striped muscles except the heart. In non-striated muscles they are never met with, and therefore the œsophagus contains them only in its upper part. They feel gritty when touched with a knife; this is due to the deposition of calcareous matter, which sometimes renders them altogether opaque, but is usually present only in their extremities, leaving their centres transparent, so that under the microscope the little worm in the interior of each of them is at once visible.

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* While these sheets are passing through the press, Dr Strachan reports cases from Jamaica.—'Brit. Med. Journ.,' June 27th, 1885.

† 'London Medical Gazette,' 1833, p. 605. Owen's paper is in the 'Zool. Trans.,' vol. i, p. 315. The oldest preparation of trichinæ is one of the sterno-hyoid muscle in the Guy's Museum, No. 1361^{30}. It was put up by Mr H. Peacock in 1828.

The earliest experiments which threw any light upon the way in which this remarkable entozoon gains access to the human body were those of Herbst, who in 1851 showed that when the flesh of animals containing trichinæ was given to other animals their flesh in turn became infested with the parasite. He did not trace the intermediate steps by which this result is brought about, and afterwards observers were for a time led astray by the notion that the trichina was a stage in the development of Trichocephalus dispar, but in the spring of 1860 Virchow and Leuckart showed that this was not the case. They fed animals with trichinous meat, and found that the worms at once escaped from their capsules and developed into sexually mature entozoa of a kind that had never before been recognised.

The experiment has since been repeated by many observers, and with uniform results. For example, an animal to which trichinous flesh has been given is killed at the end of forty-eight hours; the mucus lining its alimentary canal is found to contain numbers of minute living worms already sexually mature. They are not visible to the naked eye, but they are easily recognised under the microscope. The majority of them are females; these measure one twelfth to one ninth of an inch in length (2—3 mm.) The males are smaller, being only one eighteenth to one fourteenth of an inch long (1·2—1·6 mm.); they are further distinguished from the females by having two conical projections from the caudal extremity. If the animal be left until the sixth day before it is killed the female worms contain free embryos, which are bent and closely packed side by side in the uterus. These may even be watched under the microscope as they become extruded from the vagina and afterwards move about the field. It is estimated that at least 150 young worms are produced by each female trichina.

It is therefore clear that from the sixth or seventh day after the ingestion of trichinous flesh by man or any other animal, living trichina-embryos are poured in enormous numbers into the alimentary canal. These at once begin to bore their way through the coats of the intestine. They enter the peritoneal and other serous cavities, the lymph-glands, the viscera, and above all the muscles. Even in the heart they are often found in large numbers if the host should die or be killed within the second or third week. Observers are not yet altogether agreed as to the way in which the trichina-embryos reach distant parts of the body. Leuckart and Virchow suppose that they make their way along the connective-tissue spaces. In favour of this view it was said that they are most numerous in the muscles of the abdominal walls. But it does not appear that this is the fact, and, as Dr Thudichum points out, the rapid migration of the parasite to the most distant parts of the body is a strong argument in favour of the view that the embryos enter the blood and ultimately reach the tissues through the walls of the systemic capillaries. However this may be, it is certain that the voluntary muscles are the only parts in which they find the conditions necessary for their further development. If they enter other tissues they either perish or migrate again until they reach their proper seat. Even when they have reached the muscles they seem at first to move on in the course of the fibres, for they are found in larger numbers towards their tendinous insertions than elsewhere, as though these formed obstacles arresting their further progress. They have sometimes been distinctly seen within the sarcolemma of the primitive fibres, and Leuckart asserts that this is always their position, but others maintain that they more commonly lie between the fibres. However this may be, it is certain that they rapidly increase in size, and from being structureless and undistinguishable from minute filariæ of different species, they gradually acquire a distinct alimentary canal, and even rudimentary sexual organs. At this time they roll themselves up, and round each of them a capsule is developed. This is first to be recognised about the fourth week after their immigration. It consists originally of a nucleated transparent material, the product of the irritation of the tissues which their presence causes. After a time calcification begins in it. Dr Thudichum says that in rabbits he has seen the capsules perfectly opaque within ten weeks. But in the human subject a high degree of calcification does not occur in less than a year. Rupprecht found capsules still transparent in the muscles of a man who had had an attack of trichiniasis two years before.

Calcification of the capsule does not necessarily interfere with the life of the trichina within. The parasite remains quiescent, waiting for the death of its host, which would call forth at any moment its latent forces. But in course of time it may itself perish; it then becomes converted into an almost structureless mass, which under slight pressure breaks up into fragments.

The number of trichinæ which may be contained in the muscles of the human body is enormous. From data obtained in experiments on animals it has been estimated at from twenty to thirty millions. This, however, applies to cases which would perhaps always terminate fatally before the worms would have time to become encapsuled.

The entrance of trichina-embryos into a muscle produces certain changes in its fibres which have yet to be mentioned. They lose their striation, become brittle and homogeneous, and show numerous minute fissures. To the naked eye the muscular substance appears of a pale reddish-grey colour.

At the very time when Leuckart and Virchow were working out experimentally the life-history of the trichina, Zenker had just observed a case in which this parasite caused a fatal illness in the human subject. On January 12th, 1860, a girl was admitted into the Dresden Hospital suffering from what at first appeared to be fever. She died, and on post-mortem examination the characteristic lesions of fever were all absent, whereas the muscles were full of living trichinæ as yet unencapsuled. The girl had been in the service of a butcher, who had killed a pig about a week before her illness commenced. She had been concerned in making sausages of the pork, and had very likely eaten some of it in an uncooked state. The sausages and a ham from the pig were examined and found to contain trichinæ. It was also ascertained that the butcher and two other persons had been taken ill about the same time, but had recovered.

Since the publication of Zenker's case several others have been recorded, so that the *symptoms* of trichiniasis are now pretty thoroughly known. At first, however, they appear to be undistinguishable from those of other febrile diseases. The patient complains of loss of appetite, sleeplessness, and a sense of extreme lassitude and depression, and it may be of nausea and vomiting, but at the end of a week, or a little later, the arms and legs begin to be stiff and painful. The elbows and knees become flexed, and great pain is produced by any attempt to straighten them. After a time the limbs are sometimes rigidly extended, and the body is as though affected with opisthotonos. The muscles are tender to the touch; when grasped they feel hard and swollen, and as if they were distending the fasciæ in which they are enclosed. The jaws are often closed for several weeks, after which the muscles may become suddenly relaxed again, with an audible crack. Movements of the eyes are painful, no doubt from the presence of trichinæ in the recti and obliqui, but the power of accommodation is lost at the same time, and this is less easy of explanation. The breathing becomes shallow and hurried from implication of the thoracic muscles; and coughing, sneezing, and yawning may be almost impossible.

About the end of the second week the eyelids are observed to be œdematous, and sometimes the rest of the face and even the neck. Afterwards the legs and the parts round the joints become swollen.

The fever is not generally high in trichiniasis; the temperature seldom rises above 102° Fahr. There is often profuse sweating, and a miliary eruption may develop itself. The pulse is sometimes very rapid; a point to which some importance has been attached, as indicating that the parasites have entered the substance of the heart in large numbers. The tongue is red, slightly furred, and rather dry. The bowels are sometimes constipated; but diarrhœa may occur, especially in severe cases.

Fatal cases usually terminate in the fourth or fifth week, but sometimes much earlier. The immediate cause of death may be exhaustion, or pneumonia, or ulceration of the colon. If the patient should recover, convalescence is tardy, lasting three or four months.

The *diagnosis* of trichiniasis in well-marked cases does not appear to be difficult; the symptoms, taken together, are unlike those of any other disease. It has several times been established by the microscopical examination of a minute portion of muscle removed during the patient's life. Another way of verifying it is to search the fæces for adult worms. Rupprecht found numbers of them in the stools of patients to whom large doses of calomel had been given.

Very often, however, the discovery of the disease is facilitated by the fact that it occurs epidemically. Thus at Plauen, in 1862, thirty persons were attacked about the same time. At Hettstädt near the Hartz Mountains, four separate outbreaks of trichiniasis occurred between September, 1861, and March, 1864; in the most important of these 158 persons were attacked, of whom twenty-eight died. Other epidemics have been observed at Stassfurt, Dessau, Leipzig, and elsewhere. Of all of these full details may be found in a paper by Dr Thudichum which appeared in 1864 in the seventh Report of the Medical Officer to the Privy Council, and which contains an elaborate *résumé* of all that was then known about this subject.

In England the only cases that I know of in which trichiniasis has been detected during life, or has been proved to have been attended with symptoms, are those recorded in 1871 by Mr Dickinson, of Workington, in Cumberland. The patients were a farmer's wife, his daughter, and a serving man. They had all been eating sausages and pork from one of the farmer's home-fed pigs, the flesh of which was subsequently found by Dr Cobbold to be full of trichinæ. It has been supposed that cases in which symptoms are produced by the migration of trichina-embryos into the muscles do really occur from time to time in this country, but are overlooked. And the fact that encapsuled trichinæ are now and then found in the bodies of those who have died of other complaints, shows that the conditions which are necessary for the entrance of this parasite into the human body are not entirely absent from English life. But there is reason to believe that even in this form the parasite is rarely met with here as compared with its frequency in Germany. In Dresden, Zenker detected it in four out of 136 post-mortem examinations; and in Berlin Virchow found it six times in the course of a single year. These observers mention that often a very small number of trichinæ only were present, so that they might easily have been overlooked. Probably, in such cases the symptoms, if any, would be very slight, merely such as might be attributed to a "rheumatic affection." It is, I think, tolerably certain that, as a severe or fatal disease, trichiniasis has not yet occurred in any London hospital—at least since the publication of Zenker's case. Even on the Continent the disease is far more common in Germany than anywhere else, and most common of all in Saxony.

The severity of a case of trichiniasis appears to depend not merely upon the quantity of living trichinæ swallowed by the patient, but partly upon some unknown personal peculiarities, which favour, or interfere with, the development of the parasite. Probably, however, it varies directly with the number of embryos which penetrate into the muscles. Thus the most important guide to prognosis is believed to be the state of the patient's limbs, as regards mobility and pain on movement.

In the *treatment* of trichiniasis the first indication is to expel as many of the trichinæ as possible from the alimentary canal. For this purpose castor-oil is recommended, or calomel in twenty-grain doses, repeated at intervals. Experience is said to have shown that the latter medicine gives relief to the symptoms. Friedreich at one time proposed the picrate of potash; he gave it in one case which terminated in recovery; but live trichinæ were afterwards found in this patient's muscles. Individual symptoms must of course be treated on general principles.

The *prevention* of trichiniasis resolves itself into two distinct questions. First, how can animals intended for human food be kept free from the parasite? The answer to this is that they must be kept out of the way of eating trichinous flesh. It is indeed conceivable that adult living trichinæ, (a few of which are known to be sometimes passed in the fæces of animals in whose intestines the worm is undergoing development) might afterwards enter the alimentary canal of another animal, and that their young might migrate into its muscles. But such an explanation goes but a very little way towards accounting for the numerous numbers of encapsuled trichinæ which are often found in pigs; and they doubtless derive them from the filth which they devour. The muscles of hedgehogs, moles, and pigeons, as well as of eels, are known to be often infested with the parasite; and it has been shown to retain its vitality even after the flesh containing it has become putrid. One must not forget, therefore, that trichinæ may sometimes be derived not from pork, but from pigeons, or eels.

Secondly, if flesh containing trichinæ should by accident be used for human food, what precautions would prevent those who might eat it from having the parasite develop within their bodies? The answer is that all danger is obviated if the meat is thoroughly cooked.

An acanthocephalous or thornheaded worm, *Echinorrhynchus* sp., has only once been certainly discovered in the human intestine by Lambl ('Prager Vierteljahrschrift,' Feb., 1859). A second case, reported from Netley in 1872, is doubtful. It is common among pigs in England.*

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* In the last two sections of this chapter, which refer to Sclerostomum and Trichina, considerable additions have been made by the Editor, which it was found impracticable to mark separately.

PERITONITIS

ACUTE PERITONITIS.—*Importance—Clinical symptoms—Origin: secondary to visceral disease—Puerperal—Uræmic—Extreme rarity of idiopathic acute peritonitis—Morbid anatomy—Local peritonitis and circumscribed abscess—Diagnosis—Treatment.*

CHRONIC PERITONITIS.—*General effusion—Thickening—Adhesions—Locular effusion—Frequency—Causes.*

TUBERCULAR PERITONITIS.—*Anatomy—Symptoms—Diagnosis—Treatment.*

CANCEROUS PERITONITIS.—*Commonly secondary—Anatomy—Symptoms.*

In describing different diseases of the stomach and intestines I have repeatedly had occasion to trace their effects up to the point when, in one way or another, inflammation of the serous coat has been set up. Such inflammation very generally tends to spread over the surface of the peritoneum generally.

Acute peritonitis, the affection so produced, is exceedingly fatal; cases in which it was the immediate cause of death make up a large proportion of the total mortality from disease and injury, at least in hospital practice. Thus, taking a year at hazard, I find that in 1873, of 434 post-mortem examinations made at Guy's Hospital, in at least 52 death was directly attributable to acute peritonitis, or nearly one in eight.

The *symptoms* of this disease vary greatly in different cases, and they are often combined with and masked by those of other affections, so as to make its diagnosis difficult. The best way to give a good idea of them will be for me to take the case of a person previously supposed to be healthy, who is from some cause suddenly seized with peritonitis. Such a patient is found lying in bed with an anxions expression of countenance, features pinched and drawn, eyes sunken and often surrounded by dark areolæ. He complains of a sharp cutting or burning pain in the abdomen. This is constant, but it is liable to aggravation if he changes his posture in any way; if he coughs, or sneezes, or strains, and also when there is movement of gas in his intestines. It is also worse when he stretches out his legs in bed, so that he keeps them drawn up. As the case approaches a fatal termination the pain sometimes subsides, or even altogether ceases. It is said that this occurs especially when pus is formed in considerable quantity.

Pressure on the abdomen—and even sometimes the lightest possible application of the hand—causes much pain; the tenderness may either be diffused equally over the whole surface, or it may be most intense at some particular spot, which one may then assume to have been the starting-point of the inflammation. Even the pressure of the bedclothes may give rise to great suffering. The movements of the diaphragm and of the contents of the abdomen during the act of breathing tend to cause so much pain that the patient instinctively keeps these parts at rest, and uses the upper ribs only; the inspirations are consequently shallow and are often repeated forty or even sixty times per minute.

At the commencement of an attack of peritonitis there are generally sharp rigors. These are followed by more or less fever. The temperature may rise to 104°, or even to 105°. When death is approaching, however, the temperature falls to normal, or even below it. The hands and feet are then icy cold. The pulse is frequent, ranging from 100 to 150. At first it may be hard and strong, but it soon becomes small and feeble.

In fatal cases death usually occurs by asthenia, the mind often remaining clear to the last moment. But towards the end there may be great restlessness, the patient tossing about in all directions; he may be delirious for a few hours before the fatal termination, and sometimes this is ushered in by convulsions.

The fact that the inflammation penetrates to the subserous and muscular coats is perhaps the reason why there is almost always constipation in peritonitis, even when the inflammation did not start from any part of the intestine itself. The bowels can, however, be moved either by purgatives or enemata if the dangerous mistake is committed of interfering with them. Dr Fordyce Barker, indeed, states that in puerperal peritonitis diarrhœa is more frequent than constipation, but I do not think that this is observed in any other form of the disease.

On the other hand, sickness and vomiting are among the earliest symptoms, even although the disease may have sprung up in the lower part of the abdomen. The tongue is said to be "small, irritable, red, slightly furred, and tending to become dry." When the case is approaching a fatal termination, the patient is often tormented by powerful and obstinate hiccup. It is said that micturition is painful and difficult when the serous coat of the bladder is involved in the inflammation.

The surface of the abdomen in peritonitis is not only tender to the touch, but also much harder than natural. Its walls are distended, sometimes enormously so. The recti and other muscles are rigid, and the semilunar and transverse markings may be plainly discerned through the integuments. At first the percussion-note may be everywhere tympanitic as in health, but after a time, if inflammatory exudation should occur in large quantity, it may become dull in certain places, and the presence of fluid may be indicated by what is termed fluctuation, a sign which will be described further on. It is said that by the stethoscope effusions of lymph may sometimes be detected, a friction-sound being heard like that which I have spoken of in pleurisy (vol. i, p. 913). But I am not aware that this is ever the case in acute general peritonitis. If gas be present as well as liquid, manipulation of the abdomen may give rise to a splashing sound. Wilks mentions a case in which this was observed two or three weeks before the patient's death. In that instance it could not be satisfactorily made out whether the gas had entered the peritoneal cavity through a perforation in the intestine, or whether it had been formed during decomposition of the effused blood and inflammatory products.

Ætiology.—Acute peritonitis is most commonly caused by extension to the serous membrane of disease originating in one of the *viscera* or in the parietes of the abdomen. It would be impossible for me to enumerate in this place all the affections that may in this way set up inflammation of the peritoneum, and I do not know that a list of them would be of much practical value. As a rule, it is much less apt to follow those of the solid than those of the hollow viscera. The reason for this is that the latter, besides being capable when inflamed of undergoing perforation, and of discharging their contents into the serous cavity, are also much more subject than the former to the so-called septic forms of inflammation which, when they have reached the peritoneal surface, excite it to the same unhealthy actions. Thus cirrhosis of the liver perhaps never sets up acute general peritonitis directly; and even a hepatic abscess is much less likely to give rise to it than an ulcerating gall-bladder, the sloughing floor of an ulcer of the intestine in a case of enteric fever, or the inflamed and putrid lining of the uterus and Fallopian tubes after delivery or abortion. On the other hand, an abscess in the spleen, or sloughing infarctus (the result of heart disease), may occasionally set it up. I know of three instances in which its starting-point has been suppurative inflammation of the kidney. Other occasional causes are solid malignant tumours and even strumous enlargements of the lymph-glands.

Among diseases of the stomach the *perforating ulcer* is by far the most common cause of general peritonitis. All kinds of intestinal ulceration are apt to set it up. Even tuberculous ulcers, which are rightly said to have comparatively little tendency to perforate the serous membrane of the bowel, have done so in several cases at Guy's Hospital within the last twenty years. The various forms of *hernia* and of intestinal obstruction, again, frequently give rise to peritonitis, and so do all kinds of surgical operations on the abdominal organs.

Clinically there is a very important distinction between some of these varieties of peritonitis and others. In some of them the cause is always obvious; in others it may be utterly obscure, the patient having been supposed to be in perfect health until he is attacked by acute inflammation of the whole abdomen. The latter is frequently the case when the starting-point of the disease is a perforating ulcer of the stomach or duodenum. When it is a typhoid ulcer of the ileum, too, the occurrence of peritonitis may be the first indication of illness, for, as we have seen, enteric fever may itself be entirely latent (vol. i, p. 193). But the most important of all these obscure causes of peritonitis is the disease which I have described under the name of typhlitis (*supra*, p. 173).

Other very frequent local causes of peritonitis are affections of the internal genital organs, particularly those of the female. These may either be perfectly obvious or altogether latent.

In most, if not in all cases, *puerperal peritonitis* starts from an unhealthy inflammation of the lining membrane of the uterus which reaches the serous membrane either along the Fallopian tubes or through the tissue of the organ, the venous channels in which are often filled with pus. Miscarriages, again, are not rarely followed by peritonitis; and it may also be set up by an extra-uterine fœtation, a pelvic hæmatocele, suppuration of a Fallopian tube, sloughing of an ovary, and various other affections of the organs in question. The most careful vaginal examination should therefore be made, in every case of peritonitis the cause of which is not evident; but I have known of several instances in which it was not possible during life to determine with certainty the presence of any mischief in the uterus or ovaries, although the post-mortem examination has shown that such mischief was really the starting-point of the serous inflammation.

Acute peritonitis is not in all cases traceable to an antecedent local disease. Sir Thomas Watson, in common with many of the older writers, gave exposure to cold as one of its causes; but general pathology lends no support to such an opinion. Very strong evidence would therefore be required to convince me that peritonitis after delivery is ever set up by a chill, and does not in some way depend upon the changes that have been going on in the uterus, immediately beneath the serous surface. Dr Fordyce Barker, indeed, speaks very confidently of having seen puerperal peritonitis caused by exposure to cold; and at least shows clearly that in some cases no trace of suppuration or inflammation can be detected in the uterus or any of the neighbouring organs. But I nevertheless deem it more probable that there is really an undiscovered local cause than that these differ from all other cases of peritonitis in being set up by exposure to cold.

Again, Dr F. T. Roberts enumerates smallpox, typhoid fever, pyæmia, erysipelas, glanders, gout, rheumatic fever, &c., as so many causes of acute peritonitis. But I do not know on what facts this statement is based. Dr Wilks has recorded a case in which this disease occurred as part of an erysipelas that had started from an ulcer in the groin; but probably the inflammation then extended through the parietes, just as it sometimes does when the surface of the body is severely burnt. As regards pyæmia, the only case that I know of in which it seemed to have given rise directly to peritonitis was that of a man who died in less than an hour with cerebral symptoms, and in whom the only post-mortem appearances were, softness of the spleen, and the presence of about six ounces of pus in the lower part of the peritoneal cavity, and of a smaller quantity of pus in the left knee-joint. As no history of the case could be obtained, I had no means of knowing whether he had suffered from any symptoms of illness previously; but it was certain that he had been giving evidence in a court of law just before he was attacked with a fit, which rapidly passed into fatal coma. This case, however, was in the highest degree exceptional. Instances of ordinary pyæmia are constantly presenting themselves in the post-mortem room; and I do not know of one in which acute peritonitis was present and in which this did not start from some local mischief. I believe that at the bedside it is needless to think of pyæmia (apart from abdominal abscess) as one of the possible causes of peritonitis.

On the other hand, Bright's *disease of the kidneys* frequently causes acute peritonitis, without there being any reason to suppose that it arises by local extension of inflammation. I have notes of sixteen cases of this kind which have been observed in Guy's Hospital between the years 1854 and 1872. The inflammation was generally suppurative; and there was often a marked absence of vascular injection of the serous membrane. The kidneys were, as a rule, enlarged, and in a more or less advanced stage of epithelial nephritis; but in three cases they were contracted and granular.

I find at Guy's Hospital in twenty years only two recorded cases in which death was due to acute peritonitis, and in which the disease could not be attributed to any such cause as those of which I have been speaking. In 1874, however, several children at a school at Wandsworth were attacked at the same time with acute peritonitis. The late Dr Anstie investigated this epidemic; and the conclusion at which he arrived was that the disease was caused by exposure to the influence of sewer-gas. It was in making a post-mortem examination in one of the fatal cases that he received the wound in his finger that cost his valuable life. My friend, Dr Shirley Murphy, tells me that he has lately met with a case which appeared to be attributable to a similar cause. A woman, aged thirty-six, died on her way to the Homerton Fever Hospital. A post-mortem examination showed that acute peritonitis was the cause of death, the coils of intestine being matted together by lymph. The intestines, uterus, and other viscera were healthy, and no local starting-point for the inflammation could be discovered. It was afterwards ascertained that the patient had been living in a house the drain-pipe of which was obstructed, so that for two or three weeks the sewage had been spread over the yard, and even into the passage leading to her room. She was first taken ill on coming from a funeral; she shivered, fainted, and vomited. Next day she complained of pain in the left iliac fossa, and had purging, and two days later she died.

Anatomy.—The morbid changes which occur in the peritoneum under inflammation present in different cases considerable variations, but rather of degree than of kind. They are essentially the same as in other serous membranes; and the description will therefore, in the main, be applicable to several other diseases besides peritonitis. The surface first becomes reddened, from injection of the minute vessels. And with regard to this injection there is often, in the case of the intestines, a peculiarity. It is not uniform, but is especially marked along two longitudinal lines, which run over the bowel, at a little distance from one another, parallel with the attachment of the mesentery. The explanation of this appears to be as follows:—In health atmospheric pressure keeps every part of the serous surface in contact with some other part of it. The intestines are not (as one is apt to suppose) regularly rounded tubes; they are flattened against one another and the abdominal wall. But the distension caused by peritonitis leads them, on simple physical principles, to assume a cylindrical form; and the result is that blood is forcibly drawn into the angular spaces between them. The red lines so produced may therefore be fairly termed "suction lines," as has been proposed by Dr Moxon. They are not always present, being of course wanting whenever the intestines fail to become distended, when air has access to the peritoneal cavity, and perhaps also when inflammatory effusion is poured out very early and in large quantity.

The further morbid appearances vary according as the inflammation tends rather to the effusion of lymph or to suppuration. In the former case the membrane becomes dull and lustreless, and very soon it presents shreds and small patches of fibrin; in the latter case it is even more lustrous than in health, and to the touch it feels slightly greasy.

The inflammation seems in some cases not at any time to go beyond the formation of lymph. This forms a layer of greater or less thickness, which may either be limited to certain parts or cover the whole surface of the serous membrane. Microscopically it consists of an amorphous material, forming more or less distinct threads which cross one another in all directions, leaving interspaces in which are arranged masses of cells. The source of this material—"exudation-fibrin," as it is called, is still a matter of doubt. Virchow taught that it is not derived as such from the blood, but formed in the tissues themselves. He stated that the fluid effused into an inflamed serous cavity often contains not fibrin itself, but a substance termed fibrinogen, which passes into fibrin only when the fluid is exposed to the air, or when some other substance is added to it. Cornil and Ranvier adopt Virchow's view, and maintain that the epithelial cells of the serous membrane, modified by inflammation, become "fibrino-plastic," *i.e.* capable of converting fibrinogen into fibrin. This theory evidently involves the supposition that fluid effusion always precedes the deposition of lymph. But such a supposition is altogether opposed to the facts of morbid anatomy. Thus when inflammation of moderate intensity extends to a serous membrane from limited areas of disease in a subjacent organ, the corresponding parts of its surface become covered with local patches of lymph. And this is the case even where the affected parts are on the sloping sides of the lungs or liver, from which any fluid must necessarily have at once gravitated away. Rindfleisch also points out that granules of fibrin are often massed round the vessels in such a way as to indicate that it has oozed out of them or has been derived directly from the blood.*

The meshes of the lymph contain cells in greater or less numbers. The minute observations of Rindfleisch, Ranvier, and Klein show conclusively that some of them are derived by proliferation from the endothelium. Recent investigations have, indeed, thrown a flood of light upon the histology of serous membranes, the results of which will doubtless hereafter prove to be very important. It has been proved that stomata, very like those on the leaves of plants, exist in the endothelium and lead directly into the subjacent lymphatic vessels. Thus the tissues beneath necessarily take part in inflammatory processes affecting the serous membrane itself. Klein, in his 'Anatomy of the Lymphatic System,' speaks of the lesser omentum and mesentery being œdematous and swollen to five times their normal thickness in animals in which he had set up artificial peritonitis. The occurrence of effusion into the subserous tissues accounts for the well-known fact that in peritonitis the membrane can be stripped off much more readily than in health.

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* [Fibrin cannot ooze out of the blood: but fibrinogen no doubt does, and also leucocytes, which all recent inquiries connect more and more closely with the coagulation, *i.e.* the formation, of fibrin.—Ed.]

Whenever the inflammation goes beyond a certain degree of intensity fluid is also effused into the serous cavity. This is always somewhat turbid, and under the microscope it exhibits more or less numerous leucocytes. Modern pathology teaches that they are mainly wandering white corpuscles. When they are sufficiently abundant to give to the liquid a milky colour, this is said to be purulent. Thus every gradation may occur between the simplest form of fluid inflammatory exudation and that which is in fact pure pus. All depends upon the proportion of leucocytes to serum.

If, on the other hand, the peritonitis remains at a lower degree of intensity it leads to adhesion of the opposed surfaces of the serous membrane. The active agents by which this is brought about appear to be the cells embedded in the fibrin. Some pass into spindle-cells, and ultimately form perfect connective tissue, while others develop into blood-vessels, the walls of which are at first exceedingly soft, consisting entirely of opposed cells. These readily give way if the exudation fibrin is subjected to pressure or traction by the movements of the organ beneath. Spots of hæmorrhage are consequently often seen, and sometimes the amount of blood effused is very great. Writers have described a hæmorrhagic form of peritonitis which appears to arise in this way.

The adhesions resulting from peritonitis may be universal, the cavity being obliterated and the abdominal organs united together by connective tissue, from which they have to be dissected out when a post-mortem examination is made. More frequently, perhaps, the opposed surfaces adhere in certain places only. They still move on one another as in health; and thus the tissue which connects them becomes stretched into bands or cords which may acquire a considerable length. To these I have already referred as causes of intestinal obstruction (p. 201).

Even when the inflammation has gone on to the effusion of a large quantity of fluid, the possibility of its terminating in adhesion is by no means excluded. The fluid may be absorbed, and the two surfaces may then come together and unite. The connecting fibres seem then to be formed from the cells of a layer of granulation tissue, which covers each surface, being derived from the parenchyma of the serous membrane. Even pus may dry up and become converted into a caseous mass embedded in the substance of the adhesions.

Circumscribed peritonitis.—In most cases of peritonitis the inflammation starts from some one spot and diffuses itself over the whole abdomen. This process is doubtless much accelerated by the movements of the intestines, by which parts already inflamed are brought into contact with others which had not hitherto been reached by the disease. When the stomach or intestine has been perforated, the extravasated matters may be carried to the most distant parts of the cavity. But it by no means necessarily follows that the inflammation should thus spread over the whole surface of the peritoneum. The omentum often seems to retard for a time its progress towards the organs situated above it. And even when pus is poured out it may be limited by the agglutination of the two surfaces of the serous membrane round the space which it occupies. Thus a peritonitis starting from the uterus may lead to a circumscribed abscess occupying the pelvis and more or less of the lower part of the abdomen. I lately made autopsies in two cases of this kind, in one of which the pus had been discharged through the bladder during life, and in the other through the umbilicus. So also peritonitis arising from ulceration of the intestine often gives rise to localised collections of pus. This is especially apt to occur when the cæcum is the starting-point of the disease. The abscess then forms a swelling in the right iliac fossa. It sometimes points near the crest of the ilium, but not infrequently it passes down below Poupart's ligament and discharges in the groin. In other cases, again, it makes its way backwards towards the loin. Such abscesses are sometimes difficult to distinguish from those caused by diseased bone, and the difficulty is increased by the fact that when the crest of the ilium lies in the way of the pus, part of it sometimes becomes denuded of its periosteum so as to be within easy reach of a probe. The bowels sometimes communicate freely with an abscess of this kind, and much fæcal matter may be discharged with the pus. I have notes of one case in which for a considerable time before the patient's death almost all the fæces passed through an opening in the groin; in that instance the abscess was secondary to cancerous disease of the cæcum. The fact, however, that the pus discharged from a circumscribed abscess in the abdomen has a fæcal odour does not prove that there is a communication with the intestine. Several writers have pointed out that matter collected in the neighbourhood of the bowels may acquire such an odour as a result of the diffusion of the intestinal gases. And, on the other hand, when there is an opening this does not invariably prove that the abscess started from the ulcer. Dr Habershon has insisted on the fact that the intestine may be penetrated secondarily.

Another form of circumscribed abscess in the peritoneal cavity is limited to the sac of the lesser omentum. I have notes of one such case in which this cavity contained two or three pints of pus, the mischief having started from disease of the pancreas. Such cases are exceedingly rare. On the other hand, abscesses limited to one or other hypochondrium are by no means uncommon. In the 'Guy's Hospital Reports' for 1873-74 I have recorded several cases of this kind. In some of them the abscess started from an ulcer of the stomach, or other disease in the neighbourhood, but in most it resulted from some direct injury, particularly when seated in the right hypochondrium. I believe that in cases of the latter kind the suppuration is often preceded by a circumscribed effusion of blood, which may itself form a distinct swelling, and which is more or less closely analogous to the pelvic affection known as peri-uterine hæmatocele. One of the most interesting of my cases was that of a patient who had been kicked in the left side, and who came in with a large rounded tumour in the hypochondrium. After a time we found that air had entered it, for curious musical sounds, synchronous with the heart's beats, were heard over it, and the percussion-note became tympanitic. Yet there were no symptoms indicative of constitutional disturbance, and the patient left the hospital, refusing to believe that anything serious was the matter with him. Some time afterwards he returned, saying that he had voided a quantity of matter, and that the tumour had disappeared, and in fact no trace of it could be discovered. Cases of abscess in the hypochondrium, however, do not often terminate thus favourably.

Diagnosis.—This has to be considered from two points of view; it may either present difficulties of a *positive* or of a *negative* kind.

(1) The former arise chiefly when a person, previously supposed to be well, is suddenly seized with pain in the abdomen. It may then be far from easy to determine whether the attack is of an inflammatory nature, or whether it is colic or hysteria. The cardinal distinction is to be found in manipulation of the abdomen. In colic pressure and friction give relief; the belly, too, is often hard and contracted. The pain, too, intermits entirely from time to

time; the patient has intervals of complete ease, and when his sufferings are at their worst he is restless and tosses about in search of relief. In hysteria, on the other hand, there often appears to be the most extreme tenderness of the surface, but if the patient's attention be diverted one may find that no further complaints are made, and after a time that even considerable pressure is perfectly well borne, and that the abdominal walls are quite soft and supple. The exaggerated susceptibility and sensitiveness to the lightest touch are in such cases the very circumstances that show the absence of serious disease. One must inquire whether the patient has previously had hysterical attacks, or globus, or amenorrhœa, but it is to be borne in mind that acute peritonitis from perforating ulcer of the stomach occurs rather frequently in anæmic young women who are very likely to have had hysterical symptoms. Sir Thomas Watson remarks that a hot skin, a furred tongue, and a quick pulse are not necessarily to be regarded as proving that the pain is otherwise than simply nervous. I do not know, however, whether the thermometer ever shows the presence of fever in cases of mere hysteria, but I remember hearing of a case of colic in which some anxiety was felt for a time because the temperature was some degrees above the normal, but in which the patient was well on the following day. And, on the other hand, in peritonitis there is often no heat of skin. The probability that the pain is due to colic is of course greater if the patient has eaten any indigestible food, or if the gums present the dotted lead-line. In all doubtful cases, however, one must remember that to attribute to peritonitis a pain really due to colic is an error from which no evil consequences can follow, whereas the converse mistake may be fatal to the patient, and a few hours' delay will always solve the question.

The rupture of a concealed aneurysm into the abdominal cavity by a small aperture, or into the subperitoneal tissues, is another possible cause of sudden severe pain that must not be overlooked, particularly if the patient should have fainted when the attack began, or if he be pulseless from the first. I do not know that this can always be distinguished from perforation of the stomach or intestine.

But when one has come to the conclusion that peritonitis is present or impending, one has not completed one's diagnosis. Its *cause* remains to be discovered. Now, I have already pointed out that among the very numerous affections that may give rise to inflammation of the peritoneum, comparatively few are likely to be absolutely latent until the time the peritonitis is set up. Hence, when a person supposed to be healthy is attacked, the range of *possible*, or at least of *probable*, causes is after all not very extensive. Perforating ulcer of the stomach or duodenum, perforating typhoid ulcer of the ileum, disease of the cæcal appendix, and perhaps one or two affections of the female genitalia, are the chief among them. Now, a perforating ulcer of any part of the direct channel of the alimentary canal is commonly fatal in a few hours, or in a day or two at the latest; it may even be still more rapid. The 'Pathological Transactions' contain reports of one case in which the patient died within an hour, and of another in which death was quite sudden. The subject of it, an Oxford professor, fell down dead while walking in the streets of London. Hence I believe that where peritonitis runs a more protracted course than this in a male patient (or even in a female if the ovaries, Fallopian tubes, and uterus can be proved to be healthy), there is a very strong presumption that it started from the cæcal appendix. Even though one may at no period of the case be able to find any direct evidence in support of such a diagnosis, I think one is justified in maintaining it; but if recovery should take place, a hard mass can often be discovered in the position of the cæcum.

(2) I have now to deal with the *negative* side of the difficulty in the

diagnosis of the disease. And in illustration of this I may allude again to a case, in which acute suppurative peritonitis, starting from disease of the vermiform appendix, ran its course in a young man previously healthy, without characteristic symptoms, so that his medical advisers thought him in a fair way to recover, when they last saw him, a few hours before his death. Instances such as this are doubtless of very rare occurrence. But under other circumstances it very commonly happens that peritonitis remains altogether latent. In enteric fever, for instance, all physicians know that perforation of the intestine is often found in the post-mortem room to have taken place some hours or days before death, although there had been neither increased pain nor tenderness in the abdomen, nor, indeed, any marked aggravation of the symptoms to suggest that the fatal issue was being brought about in such a manner. The cause of this is generally supposed to be that patients suffering from fever have their senses and intelligence so greatly stupefied. But the truth is that peritonitis remains latent no less frequently in persons whose minds are clear to the last. Thus in making a post-mortem examination after an operation for hernia, ovariotomy, or the like, I have repeatedly found universal peritonitis when those who had watched the patient most closely had detected no evidence of it during life. And in any case in which symptoms of intestinal obstruction have been present for a few days, I believe that one can never assert positively that inflammation of the peritoneum may not have already set in. But then it is to be observed that the symptoms of ileus really scarcely differ at all in kind from those of peritonitis; a principal distinction being the fact that the constipation is insuperable in the one case but not in the other.

Again, in the majority of cases in which acute peritonitis is set up by Bright's disease of the kidneys, I believe that its presence is first discovered on the post-mortem table, the patient having at most complained of pain in the abdomen such as might have arisen from some trifling cause. I think that in all latent forms of peritonitis the effused fluid is generally pure pus. And in connexion with this it may be noted that some of the older writers describe the pain in peritonitis as subsiding when free suppuration has taken place. And they add that it is important for the practitioner to be aware of this, lest he should commit the error of supposing that the patient is about to recover when in reality his death is surely approaching. The most trustworthy guides to a prognosis in peritonitis are, they say, the aspect of the patient and the state of the pulse. The more frequent its beats the greater the danger.

Treatment in acute peritonitis must to a great extent be varied according to the conditions under which the disease arises. When it is nearly the last in a series of morbid changes that tend irresistibly to destroy the patient there is nothing to be gained by active interference. Very often little can be done beyond the application of hot poultices or turpentine stupes to the abdomen, and the administration of morphia by subcutaneous injections for the relief of pain.

The case is very different when the disease is set up by a perforating ulcer of the vermiform appendix or by any similar local affection. In the whole range of therapeutics there is nothing more important than the treatment of a peritonitis of this kind. It is not saying too much to assert that a single error in conducting such a case may at almost any period be the immediate cause of death; and, on the other hand, that skilful and judicious management is often the direct means of saving life. *Rest* to the inflamed parts is the one thing essential. The patient must be kept in bed from the moment that the existence of peritonitis is suspected, and must maintain the recumbent position most scrupulously, being forbidden to sit up for any purpose whatever. A pillow may be placed beneath the knees to support the thighs in a flexed position. No purgative of any kind should be administered or allowed to be given, even though the bowels should remain closed for many days; in most cases not even an enema. The importance of this rule may be made apparent by my quoting a remark of Dr Habershon's that in cases of this kind he has, at the post-mortem examination, seen castor-oil floating on the contents of the abdominal cavity.

Whether nourishment and medicines should be given by the mouth depends upon the cause to which the peritonitis seems to be attributable. When there is reason to suspect that it is due to perforating ulcer of the stomach this organ should be kept perfectly empty. A most striking instance of success from what may be termed the "starvation" plan of treatment was many years ago recorded by Dr Hughes in the 'Guy's Hospital Reports' (2nd series, vol. iv, p. 332). A young woman became collapsed, and was seized with severe pain in the stomach. The last food which she had taken was a little gruel, four hours before; for some days previously she had eaten almost nothing. She sent for Mr Ray, of Dulwich, who (instead of giving her brandy and castor-oil, as so many medical men would have done) administered only twenty minims of tincture of opium in a little water. She rallied somewhat, and was carefully removed to the hospital. She was there ordered half a grain of opium in a pill every three hours, and to have nothing whatever to drink except two measured teaspoonfuls of toast water. After two days she complained much of thirst. An enema of five ounces of strong, tepid beef tea was therefore administered, with five minims of the tincture of opium. This was afterwards repeated three times a day. She was also allowed to suck one teaspoonful of beef-tea jelly, instead of the toast water. It was not until the ninth day that she was permitted to have two tablespoonfuls of strong mutton broth. She completely recovered, and was discharged from the hospital. Nearly four months afterwards, having been so foolish as to indulge largely in cherries and gooseberries for a few days, she was attacked with the same symptoms as before. She had brandy and water given to her. She died in nineteen hours. An ulcer in the stomach was found, which had become torn away from a thick layer of old lymph by which it had before been closed. In its neighbourhood there were old vascular adhesions. I think it is certain that in this case perforation of the stomach occurred during the first attack, as well as the second, and that she would have died at that time had any less skilful plan of treatment been adopted.

In a future case of the same kind it would perhaps be well to allow the patient to have nutrient enemata from the first, so as to assuage the thirst; and to administer even the opium by the rectum. Perhaps it is never wrong to let the patient suck ice broken up into small pieces (ice *pills* as the Germans call them), but he must take them very slowly, so as hardly to make more frequent efforts to swallow the water derived from them than he ordinarily makes to get rid of the saliva which is constantly being poured into his mouth.

Unless these precautions be adopted, I believe that perforation of a gastric or duodenal ulcer, even if it should happily have taken place at a time when the stomach was empty, always terminates fatally in a few hours, or at latest within a day or two. If, therefore, one is called to a case of peritonitis which has already run on for some days, one may, in the absence of direct evidence, commonly conclude that it is due to disease of some other viscus; and very small quantities of milk, beef tea, and the like, may be allowed at frequent intervals, as the stomach may be able to bear them.

Opium should be administered freely in all cases of peritonitis. This practice was first introduced by Dr Graves, of Dublin, who, in 1822, ordered it in very large doses, to relieve the agony experienced by a woman in whom inflammation had set in after the operation for tapping. Her case seemed hopeless; but to his great astonishment she recovered. Dr Graves, however, used also to give calomel, which at the present day is believed to be unadvisable. Two grains of opium should be given at first, and afterwards one grain every two or three hours, the action of the drug being of course carefully watched. There is great tolerance of this remedy in cases of peritonitis. A lad, who has probably never swallowed a dose of opium before, will take as much as twelve grains daily, without being made sleepy or having a furred tongue, and without his pupils being in any way affected by the drug. When the disease subsides, the greatest caution should be exercised in discontinuing the remedy. I remember a case in which the bowels began to act regularly every day, while the patient was still taking a grain of opium every two hours throughout the day and night.

In many cases a few leeches may be applied to the most painful part of the abdomen with advantage; the pain is often much relieved by them. Niemeyer recommends that the whole belly should be covered with cold compresses and that these should be changed every ten minutes, or that one or more icebags should be laid upon it. I confess that my prejudices are strongly in favour of hot, rather than cold, applications. Warm fomentations or large poultices should, I think, be used constantly, being changed as often as they cool. When there is much meteorismus, great relief is often afforded by a flannel, wrung out of boiling water, and sprinkled with turpentine. A long tube, introduced into the rectum, and cautiously pushed upwards, has sometimes been known to afford escape to a large quantity of gas from the colon; but it much more often fails. If death should seem to be impending from tympanitic distension, it may be necessary to puncture the intestine, with a very fine trocar, through the parietes, but this procedure is attended with more risk than in cases of mere mechanical obstruction, because the coats of the bowel when inflamed lose their elasticity, so that the hole made by the trocar is apt to gape after its removal, and may allow fæcal matter to escape into the peritoneal cavity.

Prognosis.—It still remains that something should be said with regard to the prognosis of acute peritonitis, which, however, could not precede the consideration of its treatment. The intensely dangerous character of this disease has already been made manifest. Sometimes death is inevitable, particularly when the inflammation is set up by perforation of an ulcer in a stomach containing a considerable quantity of food. And when perforation occurs in enteric fever, recovery is doubtless exceedingly rare, although instances of it have been recorded. But as for that form of peritonitis which is set up by ulceration of the cæcal appendix, I believe that when properly treated it is infinitely less dangerous than is supposed. I have never myself seen a case of this kind terminate in death when its nature was correctly diagnosed, and when it was treated according to the rules laid down above, with no purgatives or enemata. Nor of late years have any fatal cases occurred in Guy's Hospital, except such as died very shortly after admission. I have, therefore, been accustomed to give a favourable though a guarded prognosis in cases of typhlitis, even when symptoms of diffused peritonitis are present. I am quite ready to admit that there may be cases of sloughing of the appendix, which are necessarily fatal. But, as I have shown, the differences in cases of typhlitis appear to be differences of degree, and not at all of kind; so that I believe that I am justified in saying that when this disease is skilfully treated it scarcely ever terminates otherwise than in recovery.

The peritoneum is liable not only to the acute form of inflammation which I have hitherto been describing, but also to certain chronic affections which may be either (1) *simple*, or (2) *tubercular*, or (3) *cancerous*.

Simple Chronic Peritonitis.—In this affection the whole surface of the peritoneum is thickened and opaque. Adhesions may often exist between different parts. Thus the liver, spleen, and stomach may be united into a single mass by firm connective tissue, and be closely adherent to the diaphragm and abdominal parietes. In other cases the liver is connected to the walls of the abdomen only by fibrous bands. Very frequently the omentum is drawn up, and its folds are inextricably blended together, so that with the fat which it contains it forms a solid mass, binding the colon to the stomach. The intestines may be fixed to the front wall of the abdomen; indeed, the entire peritoneal cavity may be closed by adhesions in cases of this kind. Far more commonly, however, the small intestines, if adherent at all, are so only among themselves, and they are then collected in a more or less rounded mass in front of the spine. Sometimes the membrane which unites the several coils can be stripped off, leaving the intestines still covered with a serous coat. This tendency to the formation of adventitious membranes, looking like thickenings of the peritoneal coverings of the viscera, but in reality superimposed upon them, may be seen in all parts of the abdominal cavity. Such a " reduplication " of the capsule of the liver is one of the most remarkable features of the affection known as perihepatitis; and perihepatitis, besides occurring as an independent disease, forms part of very many cases of chronic peritonitis.

The newly-formed membranes may further form adhesions among themselves, dividing the general cavity into a number of *separate chambers* each containing fluid. A very remarkable instance of this occurred in Guy's Hospital in 1860. A woman, aged forty-four, was sent to the hospital, supposed to be suffering from cystic disease of the ovaries. The physician under whom she came doubted this, and thought that there was fluid in the peritoneal cavity. After some weeks she died. At the autopsy it appeared at first as though the original diagnosis had been correct. Nothing could be seen but a mass of cysts covering the intestines and even the stomach and the liver. Presently, however, it was seen that these cysts had been formed not in any organ, but in the peritoneal cavity. Several of them lay between coils of intestine, and some contained a fluid of milky appearance, from the admixture of chyle. Another very similar case came not long ago under my own care. The abdomen contained a considerable quantity of fluid; this would have been regarded as a simple ascites (caused by the heart disease for which the patient was admitted) had it not been that the physical signs were in some respects anomalous. After death the peritoneal sac was found to be divided into a number of distinct chambers by adhesions; one of them was above the transverse colon, another occupied the middle of the abdomen, a third filled the right loin.

In the great majority of cases of chronic peritonitis, however, the small intestines are not compressed by an adventitious membrane, nor even adherent among themselves. Their coils are still capable of moving on one another, and their mesentery is fan shaped. But the mesentery is remarkably shortened. It may measure not more than about two inches from the spine to the attached edge of the bowel, which is thus tethered very closely to the back of the abdomen, instead of floating freely. Moreover, the length of the bowel itself is greatly diminished. It may not be more than a few feet long from the duodenum to the cæcum; so that the mucous membrane of the ileum is thrown into folds, resembling somewhat the valvulæ conniventes of the jejunum. Its diameter is no less contracted, so that it may hardly admit the little finger. The muscular coat of the bowel is generally thin, but that of the stomach is sometimes much thickened, so that it resembles an india-rubber bottle more than anything else (p. 156).

In the great majority of cases of chronic peritonitis, a more or less transparent straw-coloured fluid is effused into the abdominal cavity. Even when no liquid is found after death it had probably been present at a former stage of the disease. Sometimes, instead of being pale, the liquid is darkened by the presence of blood; sometimes it contains flakes of lymph or even pus; but the latter, when present, is generally the product of an acute inflammation, supervening upon the chronic disease, as a result of paracentesis. The surface of the peritoneum, besides being thickened, is opaque. But I may observe, by the way, that opacity is not a proof that chronic peritonitis had existed. In cases of dropsy the peritoneum generally, perhaps always, looks white and opaque. This has been regarded as a cadaveric change, due to imbibition by the dead tissues, but I do not see why it may not be due to the action of the fluid upon the serous membrane during life. In a large proportion of cases of chronic peritonitis, indeed, the peritoneum is not white, but blackened, or of a slaty colour, from effused blood. This appearance is often particularly marked over the intestines.

Distribution.—Simple chronic peritonitis is very far from being a rare disease. In Guy's Hospital there is on an average one case of this kind to two of ascites from cirrhosis of the liver (the most common of the local causes of ascites). It appears to be about equally frequent in both sexes. Of thirty-four cases that I have analysed, eighteen occurred in males, sixteen in females. There was in these cases a very wide range in the age of the patients. Between twenty and thirty, there were almost as many cases as between thirty and forty, or between forty and fifty; several patients were more than sixty years old, and one had passed the age of seventy.

Causes.—Very little can as yet be said of the causes of chronic peritonitis. The disease seems very seldom to be distinctly traceable to any one of the subjacent viscera; at least I find only three such cases recorded in our post-mortem books: in one it was believed to have started from the cæcum, in another from old pelvic cellulitis, and in the third from the same disease, which itself arose from morbus coxæ. Sometimes, perhaps, perihepatitis is its origin. Like that affection, it commonly occurs in patients who have Bright's disease of the kidneys, which therefore may perhaps be regarded as its principal cause. Many patients affected with chronic peritonitis have been intemperate, some have had lead-poisoning, some have been gouty, some have had disease of the heart.

The principal *symptom* of chronic peritonitis is the presence of fluid in the abdominal cavity, or *ascites,* as it is termed. This can most conveniently be discussed further on when I am describing certain other diseases which may likewise give rise to it. And I will also postpone for the present what has to be said with regard to diagnosis, prognosis, and treatment.

TUBERCULAR PERITONITIS.—In this disease the peritoneum is covered with minute grains; which, however, are seldom uniformly distributed over its surface, but are much more numerous in some parts than others, and especially on the under surface of the diaphragm and in the flanks. The serous surface of the intestines is sometimes comparatively free. The omentum often contains a large quantity of yellow cheesy material, or even of recent tubercle, and it is drawn up into a flattened mass, which may be as much as two or three inches thick, lying below the stomach and over the colon. The abdominal cavity may after death be found to contain a considerable quantity of turbid serum or even of pus, but more frequently it is closed by adhesions, or there are merely a few scattered collections of liquid here and there between the viscera. Tuberculous affections of other parts are commonly associated with tuberculous peritonitis. Thus, in women, the Fallopian tubes are almost always affected; they are much enlarged, lined with a thick caseous layer, and very often contain pus. Sometimes the same condition is present also in the cavity of the uterus. Dr Moxon thinks that the disease spreads into the open mouths of the tubes from the serous surface; he has observed that the tuberculous change is often limited to those parts of them which are furthest from the uterus. In males, again, the epididymis or testis (on one side or both) is sometimes the seat of tuberculous mischief. I have seen one case in which this was made out during life and afforded great help in the diagnosis. Other serous membranes often become affected in the same way as the peritoneum. Thus one or both of the pleural cavities may contain a considerable quantity of fluid, or they may be covered with tubercles and closed by adhesions; and I know of two cases in which tuberculous pericarditis existed, attended with the effusion of a large quantity of pus. The intestines often show tuberculous ulcers. In seven cases out of nine the lungs contain tubercles, but it very seldom happens that pulmonary disease is present in such a form as to be capable of recognition during life.

The *symptoms* of tubercular peritonitis are often very vague and obscure. The patient becomes out of health and loses flesh. He complains of pains in different parts of the abdomen. He may have diarrhœa, particularly if the intestines are ulcerated. The abdomen may be tender and harder than natural and it may feel hot. Very often it is rather retracted than enlarged, but sometimes it is tumid, and there may even be marked fluctuation, and other indications of the presence of fluid in considerable quantity. Clinically, indeed, I think that ascites is more often detected than might be supposed from mere post-mortem observations; for at an early stage of the disease the peritoneum frequently contains fluid, which is absorbed in its further progress.

Age and sex.—It would be a great mistake to suppose that tuberculous peritonitis is exclusively, or even mainly, a disease of early life. In twenty-eight successive fatal cases at Guy's Hospital, I find that two patients were under ten years of age, six between ten and twenty, eight between twenty and thirty, five between thirty and forty, three between forty and fifty, and four over fifty. The disease is more than twice as common in men as in women. Of the twenty-eight cases only eight were in females, and in all of those in whom puberty was established, with one exception, there was coexistent disease of the Fallopian tubes.

Diagnosis.—In the account which I have given of the symptoms of tubercular peritonitis there are very few points that would serve to distinguish it from other forms of chronic and subacute abdominal disease. Great assistance, therefore, is often afforded by the induration of the omentum, which may be felt as a rounded tumour running more or less obliquely across the abdomen above the umbilicus. I have known it to be mistaken for the edge of the liver depressed and rounded by thickening of its capsule, but a resonant percussion-note is elicited *above* the mass, when, if it were a hepatic tumour, there must necessarily have been absolute dulness. I remember a case in which such a tumour constituted the main clinical feature. Another sign of tubercular peritonitis is the existence of inflammation and thickening, and even of erysipelatous redness, round the umbilicus. This may perhaps sometimes result from adhesion of the small intestine to the abdominal wall at this spot; for, in one case that I know of, a fæcal fistula resulted. More commonly perhaps it is caused by an extension of the inflammation of the parietal peritoneum to the surface along the track of the obliterated umbilical vessels, just as we shall see that cancerous disease is often propagated. In some of those rare cases of strumous peritonitis in which the abdomen becomes distended with pus, the umbilicus gives way and allows the fluid to escape. The diagnosis of tubercular peritonitis may also be confirmed by the discovery of coexistent effusion into one of the pleural cavities, or into the pericardium. Lastly, whenever we suspect tubercular peritonitis in a female patient, we must not forget how constantly this disease is associated with tubercular disease of the female generative organs, of which, however, the symptoms would appear to be very vague. One patient has had amenorrhœa for eighteen months, another has had menorrhagia, a third had a miscarriage a month before her abdomen began to enlarge, a fourth had one period which lasted a fortnight, and in which the flow was excessive ; she then missed her next period, and from that date her abdomen began to swell and her fatal illness commenced.

Prognosis.—The clinical recognition of tuberculous peritonitis is the more important in that the disease has by no means so decided a tendency to terminate fatally as might be supposed. I have seen several instances in which there was reason to believe that recovery from it took place, and in one case the diagnosis was afterwards proved to be correct by a post-mortem examination. The patient (who had left the hospital apparently well) came in again some months afterwards and died with tubercles in almost all parts of his body ; and it was clear that the peritoneum had been the seat of former mischief of the same character. But the most remarkable instance that I know of, in which recovery from tuberculous peritonitis has occurred, is one recorded by Sir Spencer Wells. The patient, a female, aged twenty-two, was believed to have an ovarian tumour. She had twice been tapped, eighteen pints having on one occasion been drawn off. It was decided that ovariotomy should be performed, and Mr Wells made a simple incision. But he found that the peritoneum was studded with myriads of tubercles. Some coils of small intestine were floating, but the great mass was bound down with the colon and omentum, all nodulated with tubercles, towards the back and upper part of the abdomen. He pumped out all the fluid and closed the wound. The patient went through a sharp attack of peritonitis but got well. Four years afterwards she married, she had no children, but six years later she was stout, hearty, and well.

Treatment.—This case is the more remarkable in that no medicinal treatment appears to have been employed. For I have a decided conviction that in children tubercular peritonitis is capable in the great majority of cases of being directly cured by the local application of the linimentum hydrargyri. This practice has long been carried out in Guy's Hospital, the liniment being spread freely over the surface of a flannel belt, which is stitched tightly round the abdomen. I have more than once seen the greater part of the fluid removed within a few days under such treatment. And the patient has also improved in health and gained strength. It is true that there has been no direct proof of the tuberculous nature of the affection, but the cases in question were such as are commonly regarded as instances of "strumous peritonitis," and many of them were running a chronic course. And I believe that there is evidence to show that in children all tuberculous affections tend towards a fatal termination less uniformly than in adults. It is no doubt advisable to give cod-liver oil, syrupus ferri iodidi, and the like, but I have seen several cases in which these have failed, and in which the mercurial application has proved successful.

MALIGNANT DISEASE OF THE PERITONEUM.—This—the third chronic disease to which the serous membrane of the abdomen is liable—is of very considerable importance in several respects.

Anatomy.—It consists in the presence of an immense number of roundish or flattened granules or small tumours, with which the peritoneal surface is studded over, and which are sometimes isolated, sometimes aggregated together. Often, each little nodule is distinctly umbilicated, and it may send out processes which show a strong tendency to pucker and drag the neighbouring parts of the serous membrane towards it as a centre. In this way, as we shall presently see, the calibre of one or more of the hollow viscera may be very considerably diminished. It is probably by means of a somewhat similar process of contraction that the omentum becomes drawn up and converted into a solid mass which lies transversely across the abdomen, below the stomach. This induration of the omentum is present in most cases of malignant disease of the peritoneum. In other respects the distribution of the nodules may vary widely. Sometimes the mesentery is covered with them; in other cases it is comparatively free. Not rarely, as Dr Moxon has pointed out, the growth is far more abundant on the peritoneum lining the flanks and the diaphragm than anywhere else.

Origin and course.—Malignant disease of the peritoneum is generally spoken of as though it were a primary affection of the serous membrane. But it seldom happens that some one or other of the subjacent viscera is not the seat of a similar growth; and to this, I think, that in all probability the peritoneal affection is really secondary.

As Virchow pointed out, the organs most frequently concerned are the *stomach* and *ovaries*. Out of forty-five consecutive cases of extensive malignant disease of the peritoneum that have occurred at Guy's Hospital, I find that in only six are all the viscera stated to have been free from such disease. In nineteen the ovaries were affected, being often converted into large tumours by the growth. In seventeen the stomach was diseased in the same way; seven times without, and ten times with, malignant disease of the ovaries. In three cases the peritoneal affection appeared to have started from the *uterus*, in two from the *rectum*, in three from the neighbourhood of the *pancreas*. Even these facts fail to indicate the full frequency with which the pelvic organs are diseased in cases of this kind. In two there was a hard mass in front of the rectum; and in several cases (including some of those in which the disease seemed to have commenced in the ovaries) the uterus and its appendages were matted together and fixed to the adjacent parts by a large diffused growth in the subperitoneal tissue.

The way in which malignant disease spreads from the stomach or ovaries over the peritoneal surface generally is a matter of much interest. When such disease reaches the serous surface of an organ, it is well known to be capable of infecting the surface opposed to it, without the formation of adhesions between them. I lately met with an excellent instance of this. The uterus, in a case which I examined, had its body affected with cancer, which reached its outer surface. The omentum was long and hung down into the pelvis so as to touch the uterus, and in its extreme lower end there was a hard mass, resembling the uterine cancer exactly in its character. There was no malignant growth in any other part of the peritoneum. It is very probable that such local infection of the omentum is really not uncommon, and forms the starting-point of the remarkable change in this structure which I have already mentioned. The infection of the general surface of the serous membrane probably arises in the course of the movements of the contained organs. It is even possible, to use the words of Rindfleisch, that "the mutual friction of the viscera may detach bits of the nodules, and carry them hither and thither over the smooth surface of the membrane, until they find their way into some fold or recess, when they give rise to the development of fresh nodules."

Histology.—The microscopical characters and real structure of the malignant nodules doubtless vary in different cases. Virchow appears to be disposed to include a considerable proportion of them among the sarcomata, but most other writers speak of them as "cancerous" in the strictest sense of the word. However, they generally yield but little juice from their cut surfaces; and their structure is to a large extent fibrous.

Sex and age.—Unlike tubercular peritonitis, cancer of the peritoneum appears to occur much more frequently in women than in men. I find that out of the forty-five fatal cases referred to above, only eleven occurred in males. Below the age of thirty this disease is exceedingly rare. Between thirty and forty it is not very uncommon in women, but is very seldom seen in men. In each sex the most numerous cases occur between the ages of fifty and sixty; it is also common between sixty and seventy, and in one instance it was found in a man who died at the age of eighty-two.

Diagnosis.—Clinically, malignant disease of the peritoneum presents itself in different cases with very different symptoms. The growth may, by the contraction and puckering which it causes, so narrow the intestine as to interfere with the passage of its contents, and even to give rise to well-marked ileus. I have already had occasion to refer to this at p. 199. Much more frequently, however, the most marked effect of the disease is what is termed ascites; the presence of fluid in the serous cavity. I shall hereafter have to discuss the grounds of diagnosis between the various affections which may give rise to this symptom. But I may remark in this place that the induration and nodulation of the omentum in malignant disease can sometimes be made out by palpation; or a cancerous tumour of the ovary or pylorus may be felt. Another character, which I believe to have been first pointed out by Sir William Jenner, is the occasional presence of a hard mass in the skin and other tissues round the umbilicus. This I have myself observed in one case, and I suppose that the growth travels along the connective tissue in the path of the obliterated umbilical vessels.

I mentioned above that in simple chronic peritonitis the fluid poured out into the abdominal cavity was sometimes discoloured by blood. This is, however, much more frequently the case when the serous membrane is the seat of malignant disease. The fluid is then very generally of a red or brown colour; indeed, so much hæmorrhage sometimes takes place that coagula of considerable size are formed.

In other instances, again, the principal symptom of malignant disease of the peritoneum is an increase in size of the abdomen, without any fluid being present. In these cases the growth is a true carcinoma, which has undergone colloid degeneration. All the organs may be enveloped in thick layers of this substance, in the form of round gelatinous masses, many of which are attached only by the most delicate threads, or even seem to be entirely free. Many years ago, when I was a senior student at the hospital, a medical man in the country asked me, during the vacation, to look at a case in which he was about to tap the peritoneal cavity for ascites. I found that although there was very great enlargement and dulness on percussion over the whole abdomen, yet no fluctuation could anywhere be discovered. I happened to remember hearing Dr Wilks describe colloid cancer of the peritoneum, and ventured to suggest that the case was one of this kind, and that paracentesis would lead to no result. During a subsequent vacation I made the autopsy, and found that I had been right. Such cases, however, are very rare. In one instance, which occurred at Guy's Hospital in 1861, the affection of the peritoneum seemed to have started from a cancerous growth in the ascending colon. The omentum formed a solid mass an inch thick, which was spread over the intestines, and reached down to the pubes.

DISEASES OF THE LIVER

FUNCTIONAL DISORDERS—HEPATIC DYSPEPSIA

Distinctive characters of bilious or hepatic dyspepsia—Independent of secretion of bile—Murchison's theory—Formation of urea—Lithæmia—Symptoms of hepatic dyspepsia—Treatment.

In a former chapter I have discussed in detail the symptoms of what may be termed primary dyspepsia, indigestion arising from faulty action on the part of the stomach and duodenum. I then purposely omitted from my description certain other symptoms which are very commonly associated with those that I mentioned, but may also occur independently.

Besides their complaints of distension and tightness in the epigastrium after meals, &c., dyspeptic persons may experience a dull aching in the right hypochondrium, and sometimes occasional shooting pains in the same region. They have a sense of weight and fulness below the right ribs, which is often greatly increased by lying on the left side, and is also worse after meals. The hepatic region may even be tender to pressure. Another pain, of which such patients often complain, is situated in the right shoulder; or sometimes in the left shoulder. The conjunctivæ have generally a slight yellow tint, and the skin may even display traces of the same colour. The urine is generally scanty and high coloured, and as it cools often deposits large quantities of lithates of a bright red colour. The bowels are confined, and there is a tendency to hæmorrhoids.

Now, there is considerable difficulty in defining precisely the cause of this group of symptoms, but it can hardly be doubted that they depend in some way upon an imperfect functional activity on the part of the liver.

It is, in the first place, to be noted that such complaints are especially apt to occur in persons of the middle period of life, above the age of thirty-five. Those persons are particularly liable to them who take but little exercise, and who eat or drink to excess. On the other hand they are often at once removed by a few days' shooting or hunting, or by any other kind of bodily exercise which possesses sufficient attractions to induce men to give up their ordinary sedentary habits.

Probably most people belonging to the richer classes, who live in cities, take more food than they require, and many of those who live in the country do the same. Men get into certain ways as regards diet when they are young or when they are leading active lives; but later on, when their circumstances are altered, they are apt to forget that their habits ought to be altered likewise. Some kinds of food are much more apt than others to produce the symptoms I have been describing. The most injurious are generally fatty and saccharine matters, and certain alcoholic drinks, especially those which contain much sugar. All malt liquors are apt to disagree with people who suffer in this way, but particularly porter and the stronger kinds of sweet ale. Among wines, port, Madeira, champagne, and dark sherry are especially harmful; and brandy and sweet liqueurs among spirits. The evil effects are more marked in hot climates, and in the warmer seasons of the year, than under opposite conditions.

Now, non-professional persons are wont to speak of the symptoms under consideration as indicative of a "torpid state" of the liver, and to think that they are caused by a deficiency in the amount of bile secreted by that organ. The correctness of this opinion was formerly supposed to be established by the fact that the complaint is often easily removed, at least for a time, by certain medicines (particularly mercurials) which at the same time bring away from the bowels a considerable quantity of semifluid or fluid fæces, apparently loaded with bile. Such an explanation was once accepted by the faculty with as much confidence as by the outside public; and indeed it was doubtless originally promulgated by the medical authorities of a past age. But for many years past physicians have been aware that there are great difficulties in the way of its acceptance. In the first place, a large number of experiments have been made to determine whether mercury and the other drugs above alluded to possess the power of increasing the amount of bile secreted, and the general result has been that no such power has been discovered. It is true that we cannot from the action of a drug on one man who is in health safely infer what will be the action of that drug on another man when ill; still less from its effects when given to healthy animals what will be its effects when administered to diseased men. But notwithstanding that objections of this kind have been addressed to the experimental physiologists who have worked at the subject medical men are now pretty well agreed to discard the theory which attributes merely to defective secretion of bile the symptoms above enumerated, and to augmented formation of bile their cure by mercury and similar agents.

Another way of explaining the complaint is to attribute it to congestion of the portal system, and particularly of the liver. And the remedies for it are those supposed to act by emptying the overloaded blood-vessels. It is indeed well known to physiologists that the act of digestion always causes an augmented flow of blood through the liver; and of course all those elements of the food which are absorbed by the venous radioles pass directly into that organ. Hence it is certainly not a far-fetched hypothesis to suppose that the liver becomes congested in people who eat and drink too much, or when food is of too rich a kind.

There are, however, certain facts of the case which this theory leaves altogether unexplained, particularly the presence of excessive quantities of urates in the urine of these patients, and the superiority of mercurials and similar medicines over other drugs which have equally purgative properties and which ought therefore to be no less efficacious, if the indication for treatment were the merely mechanical one of relieving congestion.

Dr Murchison in his Croonian Lectures (1874) supplemented the ancient theories upon this subject by referring to certain facts which appear to show that the healthy liver plays a very important and perhaps the principal part in carrying on those chemical changes by which albuminous substances are disintegrated in the body, and which normally result in the production of urea. Some of these facts have been acquired by physiological experiments upon animals, but others are pathological, particularly the remarkable fact that urea* is absent from the urine in yellow atrophy of the liver, in which disease the hepatic cells are destroyed.

Murchison's suggestion, therefore, is that in the cases now under consideration there is not only a defective secretion of bile, but also an interference with the normal processes by which albumen is disintegrated in the liver. The result is that instead of urea (CH_4N_2O) uric acid ($C_5H_4N_4O_3$), a less perfectly oxidised body, is formed. He supposes that mercury and similar remedies have a double action. On the one hand, whether or not they increase the amount of bile secreted by the liver, he points out that they certainly increase the quantity which is eliminated from the bowels. Less of the biliary ingredients are reabsorbed and the result is that the blood is freed from their presence in excess. On the other hand, he imagines that the drugs in question may have a special power of promoting or in some way influencing the disintegration of albumen.

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* It ought in fairness to be mentioned that the chlorides and sulphates are also wanting from the urine in acute yellow atrophy, although the liver can hardly be concerned in their disappearance.

It is important to observe that the significance of these views is by no means limited to the class of cases which we have hitherto been considering. On the contrary, the substitution of uric acid for urea as the final product of disintegration of albuminous substances within the body is a most important matter. The comparative insolubility of this body and of its salts, prevents its being readily excreted by the kidneys like urea. Hence, it accumulates in the blood, and a condition arises which Murchison proposes to term *lithæmia*. The urate of soda is very apt to crystallize out into the cartilages of the joints and elsewhere; and this is *gout*. Again, even when the uric acid has been taken from the blood by the kidney it is often deposited from the urine either in the renal pelvis or in the bladder. We have then the common form of *gravel* and those important varieties of *calculus* of which uric acid and urates are the main ingredients.

I propose, in a future chapter, to continue this branch of my subject so far as concerns the disease which is called gout. But at present I must complete what I have to say concerning those slighter and less serious effects of lithæmia which, as we have seen, are commonly attributed to "torpor" or "congestion" of the Liver.

Now, as Murchison has pointed out, besides the symptoms of this complaint which have been already enumerated many others are met with which are of a less well-known kind. One of them is a bitter or "coppery" taste in the mouth, especially in the morning; this, he suggests, may possibly be due to the presence in the blood of tauro-cholic acid. Another is intestinal hæmorrhage. This he has known to occur repeatedly in the same person at intervals of many years; usually in patients beyond middle age. Each attack is usually preceded by a feeling of oppression and heaviness, or by creeping sensations, and more rarely severe neuralgic pains, suggesting the presence of gall-stones. Aching pains in the limbs, again, and lassitude, coming on about an hour after a full meal, are not seldom complained of by persons affected with lithæmia, and sometimes they are accompanied by an irresistible drowsiness. Severe cramps in the legs and in different parts of the body may be another indication of the same condition. Murchison says that they often come on during the night and are most common in cold and damp weather. He quotes from Bence Jones two remarkable instances.

The first occurred in a gentleman, aged forty, who for years had constantly had deposits of lithic acid and lithates in the urine. He then became subject to attacks of violent pain in the stomach, coming on from one to five hours after a late dinner. The pain was intermittently spasmodic; its greatest intensity was reached in half a minute, it then relaxed to return as badly as before in two minutes. When about an hour had passed the suffering gradually subsided, leaving a tenderness on pressure and an irritability after food for two or three days. After the attack the urine always deposited crystals of uric acid. The complaint had lasted for several months, but under care in diet and the use of alkalies the pain entirely ceased to recur.

The second case is that of a patient, also the subject of lithæmia, who was seized with violent cramps in the rectum, coming on six or eight hours after food, and lasting from half an hour to an hour. The same treatment which was employed in the first case was completely successful in this case also.

Another frequent symptom of hepatic disorder is *headache.* This is described by Murchison as being most commonly a dull heavy pain, seated in the forehead or more rarely in the occiput. It generally comes on when the patient first wakes in the morning, and it may either quickly pass off or last the greater part of the day, or even for several days. This kind of headache usually follows some indiscretion in diet or is preceded by constipation of the bowels. It is particularly to be noted that what are commonly called *bilious* or *sick* headaches are of a different character. They have been described among the diseases of the nervous system, under the name of migraine, which is essentially a neurosis (vol. i, p. 667). Still one can by no means deny that attacks of it in the most typical form are sometimes in very large part due to lithæmia. Dr Liveing mentions a family in which the father had gout, his son migraine, and his son again gout. And Trousseau speaks of having seen migraine and gout alternate in the same patient, an attack of gouty inflammation in certain joints being followed by the cessation of sick headaches which had previously been of frequent occurrence.

It will be observed that the complaints to which I am now referring are those which were formerly ascribed to "suppressed" or "latent" gout. And one of the advantages offered by Murchison's term lithæmia is that it renders these phrases superfluous. Another of the effects of this condition is giddiness, which, according to Dr Wilks, presents certain special characters. He says that, if due to digestive disorder, swimming in the head is especially apt to come on when the patient stoops or lays his head upon the pillow, and that it often passes off when he assumes the erect posture. But in speaking of vertigo as a neurosis I endeavoured to show that this is too narrow a view of the matter (*cf.* vol. i, p. 699).

Another symptom, which was first described by Graves, and which appears to be caused by lithæmia, is a tendency to grind the teeth. He has related some remarkable cases of this kind. The affection is described as depending upon a disagreeable uneasy sensation referred to the teeth, and relieved for the moment by the act of forcibly grinding them together. In one case, that of the Countess of E—, this habit was so confirmed that she practised it all day and consequently was obliged to give up society for several years before her death. The teeth became worn down to their sockets. During sleep the grinding entirely ceased so that the affection was altogether different from that which is so common a symptom of irritation of the brain and especially of tubercular meningitis. Graves states that all the cases he had seen have been in patients of confirmed gouty habit.* He was never able to discover any means of alleviating this troublesome complaint.

Again, it appears that *convulsive attacks* may sometimes be due to the same cause. Murchison relates the case of a gentleman who had long suffered from hepatic derangement, and who became subject to severe spasmodic twitchings in his legs. These were followed on three occasions by epileptiform seizures. A little later he had a first attack of gout, and afterwards he suffered frequently from that disease, but he had no return of the convulsions or muscular twitchings. *Noises in the ears* may also be among the effects of lithæmia. One patient, says Murchison, has the feeling of a strong wind blowing into his ear; another compares the noise to that of flowing water or describes it as a singing or buzzing; while in yet another the sound pulsates with the beats of the heart.

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* Some observers have supposed that the mere fact of the teeth being ground down, so as to show the dentine in section, is to be regarded as evidence of a gouty diathesis, or (as I should prefer to call it) a condition of lithæmia. My colleague, Mr Moon, however, tells me that dentists are acquainted with several distinct causes which may lead to this condition. One of these is that formation of the jaws which gives what is termed an edge-bite; another is the absence of enamel at the summits of the teeth.

Then, again, there may be *sleeplessness.* As has already been stated, lithæmic patients are often heavy and drowsy after a full meal and fall asleep at once when they go to bed, but after three or four hours they often awake, and they may then lie awake for hours or keep dozing off, and waking again after unquiet dreams. Murchison says that harm is often done to such patients by the administration of soporifics, and that some of them have told him that they never sleep so well as after a dose of calomel or blue pill. Depression of spirits and irritability of temper are well known to be frequent effects of the same cause.

Lastly, Murchison enumerates palpitations and flutterings of the heart, exaggerated pulsation of the large arteries, irregularity and intermission of the pulse; and, as concerns the organs of respiration, chronic catarrh of the fauces, chronic bronchitis and spasmodic asthma.

Treatment.—I have quoted in detail these cases and remarks of Murchison's because I believe that at the present day complaints of this nature are very apt to be misunderstood or neglected. It is no doubt true that formerly the opposite error was almost universal, and that many cases of headache, giddiness, and dimness of sight, which are now easily cured by quinine and similar remedies, were then submitted to severe mercurial and "alterative" treatment with injurious results. But of late years that kind of practice has fallen into disuse. The discussions and doubts that have prevailed as to the way in which mercury acts have, I believe, had very much to do in bringing about this result. The younger school of physicians, unable to prove that the metal is capable of increasing the amount of bile secreted by the liver, have forgotten that this after all is not the whole question. Hence they have become accustomed to ignore altogether the class of cases to which I have been referring, and have given up the use of remedies by which such cases are generally readily and safely relieved. For it is unquestionable that three or four grains of blue pill, with as much of the compound colocynth pill, or followed (if necessary) by the traditional haustus sennæ or some less nauseous aperient, do great good in cases of "torpid" liver. And I believe that an occasional dose of this kind does no harm whatever, except that it is apt to leave the bowels somewhat constipated, so that the purgative has afterwards to be repeated. Podophyllin has been very much recommended for cases of this kind. But the slowness and uncertainty of its action constitute great objections to its use. According to Murchison, an emetic is sometimes useful at the commencement of the treatment.

The best remedies for the condition which we are now considering appear to be certain German mineral waters, particularly those of Püllna, Friedrichshalle, and Carlsbad, all of which contain sulphate of soda, and the former two sulphate of magnesia also. The proper dose is about five ounces of Püllna water, seven ounces of Friedrichshalle water, eight or ten ounces of Carlsbad water. The necessary quantity should be mixed with a little hot water and taken the first thing in the morning or about an hour before breakfast. It secures a free full action of the bowels, and with this advantage over ordinary purgatives, that there is no constipation afterwards, nor does the dose require to be increased in the course of time. On the contrary, it is said that the quantity taken may be gradually reduced without any diminution of its efficacy. After a course of about six or eight weeks the remedy may be generally omitted, at least for a time, but patients sometimes continue to take it regularly for four or five years.*

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* See an admirable lecture by Sir Henry Thompson in the 'Lancet' for Jan. 13th, 1872.

At the same time, regulation of the diet is of the utmost importance, particularly as regards alcoholic liquids. The stronger wines, champagne, and malt liquors should be prohibited; and the patient should be limited to a very moderate allowance of a light sound Bordeaux or Rhine wine with his principal meals, or of brandy or whisky diluted largely with water.

In most cases of this kind, the mineral acids (particularly nitro-hydrochloric acid), with taraxacum and gentian, appear to be very serviceable.

Sometimes also recourse may be had to the nitro-hydrochloric acid bath so strongly advocated by Sir Ranald Martin. It is made by mixing two ounces of strong hydrochloric acid and one ounce of strong nitric acid with two gallons of water in a glazed earthen or wooden vessel, at a temperature of 96° to 98°. The feet only are placed in the bath, and the inside of the legs and thighs, the right hypochondrium and insides of the arms are sponged with the liquid. Or the abdomen may be swathed in flannels soaked with it. The process is repeated each night and morning for half an hour at a time.

The patient should also be made to take a fair amount of exercise. [Of all kinds horse-exercise is the best. Rowing is also excellent, and any exertion which produces deep breathing and free sweating. Walking, however good in other ways, is perhaps least useful for this form of hepatic dyspepsia. A quarter of an hour's game at rackets, for instance, is far more useful to most persons than an hour's walk. This advice applies particularly to persons much engaged in business which keeps them on their legs all day. A long walk only makes them more tired and unable to digest the heavy meal which their sense of exhaustion prompts them to eat; whereas a much shorter time spent in riding or rowing exercises the whole body, and after a short rest, or if needful, a quarter of an hour's sleep, they can enjoy dinner with a zest "that after no repenting draws."—Ed.]

JAUNDICE

AND THE DISEASES WHICH PRODUCE IT

Symptoms of jaundice—Tests for bile in the urine—Simple jaundice—Its course, pathology and prognosis.

Acute Yellow Atrophy.—*Anatomy—Symptoms—Event—Pathology.*

Febrile jaundice—Pylephlebitis—Jaundice from cirrhosis—from gall-stones—from cancer of the ducts—from tumours, &c.

Effects of permanent jaundice on the liver—Pathological theories of jaundice—Effects on the bowels and the skin—Pruritus—Xanthelasma.

Treatment of simple jaundice—of acute atrophy of the liver—of gall-stones—of permanent jaundice.

Unlike other secretions, bile has a colour so intense that it is capable of staining the solid tissues. Whenever the colouring matter of the bile is formed within the body, but fails to escape by the natural passages, it is carried into the general circulation by the blood of the hepatic veins. It is then deposited in the skin and other parts, and the patient acquires a yellow colour. This constitutes *jaundice*, or (if we prefer a less common term) *icterus*. The first name is derived from the French word *jaune*, "yellow;" the second is Greek for the golden thrush, the sight of which by a jaundiced person was in ancient times believed to be followed by death to the bird, but recovery to the patient.

Now, a large number of the diseases of the liver are attended with jaundice; but by no means all of them. Still there appears to be no other way of approaching this part of my subject so good as that of considering the characters of the symptom in question and the way in which it is produced. For I must especially insist upon the fact that jaundice is itself no disease due to a single cause, nor capable of being treated with drugs without further investigation of its mode of origin. On the contrary, it is one of the best examples that can be given of a morbid state, which appears an entity to the unprofessional public, but which we know to require a full analysis in every instance before we can form any judgment as to its nature.

General symptoms.—The parts that most obviously display the yellow colour are of course those which are most exposed to view, and which at the same time are naturally either colourless or at least pale. Thus, even in the skin, the redder parts are far less decidedly altered in appearance than those which are less florid. And in the superficial mucous membranes a similar difference prevails and is very striking. For, whereas even in extreme degrees of jaundice the lips and lining of the cheeks show comparatively little change, the yellow hue is exceedingly well marked in the conjunctivæ, through which in health the pearly-white sclerotic beneath is plainly visible. Indeed, the natural whiteness of the eyeballs enables us to recognise in them slight alterations of colour which are quite inappreciable elsewhere. Just as we place a sheet of white paper beneath any transparent substance, the colour of which we desire to scrutinise, so we always look to the conjunctivæ for the first signs of impending icterus, or for the faint indications of retention of bile-pigment too slight to produce general jaundice.

Whenever jaundice is present in any considerable degree of intensity, the internal parts are affected as well as those on the surface of the body. All the paler mucous membranes have a marked yellow colour. The same is true of the connective tissue generally and of the serous membranes; and any collections of fluid within the serous cavities are much more yellow than under other conditions. The lungs and the kidneys, and almost all other organs which are not so red as to conceal their yellow tint, are evidently jaundiced. The liver displays the same colour, often in an extreme degree. It has been said that the brain participates in this change, which its natural whiteness would of course make very evident. But my own observations on this point confirm those of Dr Moxon, who has asserted that the brain shows no abnormal colour, even in advanced jaundice. The transparent humours of the eye seem sometimes to be yellow and sometimes not.

The colour of the skin in jaundice varies according to its intensity and its duration. If the natural escape of the bile be suddenly completely arrested, the body may quickly assume a deep orange hue. But in many cases this is more gradually developed, the tint being at first a pale sulphur yellow. The whole cutaneous surface does not necessarily present the same depth of jaundice; this is generally more marked on the face and upper limbs than in the lower part of the body and in the legs. When jaundice has existed for a considerable length of time, the colour frequently undergoes a change; it is no longer yellow, but becomes greenish, and after a time passes into a dark olive colour. This change doubtless corresponds with the alteration which bile itself always undergoes when exposed to the air in assuming a green colour. Chemists have shown this to be due to the conversion of bilirubin (the principal colouring matter of the bile) into another principle named biliverdin, and biliverdin itself is not a very stable compound, but after a time turns brown, passing into what is called choletelin. Persons in whom the skin assumed the dark green colour above referred to were formerly said to have "black jaundice," and it was supposed to be an indication that the hepatic disease was of a cancerous nature. We now know that it means only that the jaundice has lasted for a long time, but it is true that such cases are generally cancerous. I believe that for the production of a green or "black" tint in jaundice it is necessary that the flow of bile through the ducts should be *completely* arrested. If this be not the case, I find that the skin still remains yellow, however long the jaundice may last. But before I can discuss these points fully, I must first describe in detail the principal diseases of the liver in which jaundice occurs; *i.e.* the characteristics of the chief clinical varieties of jaundice.

It may well be supposed that there is seldom much difficulty in discovering whether a patient is or is not jaundiced. Only a very careless or inexperienced physician would mistake for it the greenish-yellow hue of chlorosis, or the yellowish waxen tint commonly seen in cancerous diseases of the abdominal viscera, or the dusky sallow look of those who have had malarious fever. Nor at the present time ought anyone to confound with the olive-green tint of "black" jaundice the brown or bronze colour of suprarenal disease, although it must be remembered that until this disease was discovered by Addison, patients affected with it were commonly supposed to be jaundiced. In all the conditions just mentioned, but particularly in the last, the conjunctivæ retain their natural pearly-white appearance. One thing which must be borne in mind is that the yellow tint of jaundice is quite imperceptible by gas- or candle-light. Thus its presence may be overlooked altogether if the patient is not seen in the daytime. Cases are, however, sometimes met with in which it is difficult to be sure whether partial jaundice is or is not present. I believe that the doubt then generally lies between jaundice and the idiopathic (so-called pernicious) anæmia. In that disease the conjunctivæ often look yellowish, but this (as

Murchison has pointed out) is really caused by the presence of small masses of fat in the submucous tissue. The yellow colour is only partial, instead of being uniformly distributed over the whole surface of the conjunctivæ.

Several of the secretions of the body contain biliary colouring matter in jaundice. The sweat is yellow, so that the patient's linen is often much stained under the armpits and at those parts where there has been much perspiration. The milk has been noticed to be yellow by Bright and others. On the other hand, the secretions of the various mucous membranes remain free from bile-pigment. The saliva is colourless, and although the patient not unfrequently complains that he has a bitter taste in his mouth, there is, I believe, no reason to suppose that this is ever due to the actual presence of bile in any of the fluids poured into the buccal cavity. Even the gall-bladder and the ducts of the liver itself (as we shall presently see) pour out a colourless mucus. That the secretions of the intestinal mucous membrane contain no bile-pigment is evident from the fact that the fæces are commonly of a greyish-white colour, or (to use the common expression) "clay coloured."

Wherever any diffusible substance is present in the blood in quantity larger than natural its removal is effected by the kidneys rather than by any other organ. Hence we might expect that in jaundice the urine would contain more of the biliary colouring matter than any other secretion. The colour of the urine in cases of this kind, however, may vary widely, from a yellow, scarcely deeper than natural, to a dark brown, a greenish brown, or even a black, so intense that one can recognise its colour only by looking at the margin of the fluid, or pouring some of it out in a thin layer. The presence of bile-pigment in the urine is, I believe, a necessary part of jaundice. There is, indeed, one condition in which the secretion may for a short time have its natural appearance, although the patient's skin is still of a deep yellow colour. This is when the cause of the jaundice has been suddenly removed, particularly if it has lasted for some time. The bile-pigment then ceases to circulate in the blood, and the kidneys no longer excrete it in any quantity. But the skin does not at once give up all the colouring matter that had been deposited in its tissues, and it remains for a few days yellow. So far as I know this is the single exception to the rule that the urine invariably contains the colouring matter of bile when there is jaundice. Indeed, that secretion appears to afford more delicate indications than even the conjunctiva of the fact that the circulating fluid contains bile-pigment which has failed to find its way out of the body through the natural passages. When jaundice is commencing, the colouring matter may be detected in the urine before any of the solid tissues betray its presence, and when the blood is but slightly charged with bile-pigment this may be excreted by the kidneys, so as to be present in an appreciable quantity in the urine without even the conjunctiva affording any indication of it.

Test for bilirubin.—But the presence of bile-pigment in the urine is not to be assumed from the colour of the liquid alone. We have a chemical test which is capable of detecting it even in the most minute proportion. This is commonly known in Germany as Gmelin's test. It consists in the addition of fuming red nitric acid (containing nitrous acid) to a small quantity of the urine. This causes a beautiful play of colour if bile-pigment be present. A very good way of employing the reagent is to pour a drop or two of the urine on the flat surface of a white plate, and then carefully to add to it a single drop of the nitric acid. Round the drop of acid a series of colours are developed, rapidly passing through the shades of green, blue, and violet, into red, and finally becoming a dirty yellow. Neubauer and Vogel recommended, for the detection of small quantities of bile-pigment in urine, that the nitric acid (which must not contain too much nitrous acid) should be poured about an inch high into a conical glass, and that a little of the urine should then be carefully spread over its surface by means of a pipette. The play of colour commences at the line where the fluids come into contact with a beautiful green ring, which gradually extends upwards, and at its under surface exhibits a blue, violet, red, and lastly a yellow ring. The most recent writers are agreed that in this experiment the green colouration is the only part of the reaction which affords evidence of the presence of bile-pigment. Frerichs formerly taught that this colouring matter is liable to undergo changes which might reduce the action of nitric acid upon it to the mere production of a red or dark bluish-red colour. But Murchison observes that he has frequently found the urine to give such colours as these when there was no jaundice, and I think every physician must have made the same observation.

In fact, it is now well known that when nitric acid is added to urine in the way described above, red and violet rings may be produced by another substance, of which a small quantity is present even in healthy urine, and which is found in considerable amounts under various pathological conditions. This was once called uroxanthin, but a better name for it is *indican*, for it is the same principle which, when obtained from the indigo plant, has long been known as the mother substance of the indigo pigments. Of late years many observers have endeavoured to attach a clinical significance to the presence of indican in excessive quantity in the urine, but their labours have not as yet been successful, and therefore it will not be necessary for me to make any further allusion to this substance.

The most delicate of all methods of applying Gmelin's test for bile-pigment is to shake large quantities of the urine successively with chloroform. This extracts any of the pigment that may be present, and when nitric acid is afterwards spread over the chloroform, the reaction takes place (it is said) in a very beautiful form.

Test for bile-acids.—Hitherto, in speaking of the retention of bile within the body, and of its failing to be evacuated through the natural passages, I have alluded to the colouring matter only. I have avoided all reference to the biliary acids. Now, there has been considerable difference of opinion with regard to the question whether these acids are excreted by the kidneys in jaundice, and particularly whether this is the case in some forms of jaundice and not in others.

Unfortunately it happens that the principal chemical test for the biliary acids—that which is known as Pettenkofer's test—is both difficult and uncertain in its application to the urine. The test itself consists in the admixture of a few drops of syrup, or a few grains of sugar, with the liquid suspected to contain biliary acids, and the subsequent addition of strong sulphuric acid, precautions being taken to prevent the development of too great heat. A beautiful violet colour appears if the acids of the bile are present. Now, most authorities are agreed that Pettenkofer's test cannot be satisfactorily applied to urine unless the urinary pigments are first separated, for if this be not done the sulphuric acid chars and blackens the liquid, so that no violet colour can be seen. But Dr Harley recommends the use of the test without the adoption of any such precautions. It is sufficient, he says, to pour the sulphuric acid into the urine without mixing the two liquids, and at the line of contact a deep purple hue may after a few minutes be observed. Murchison, however, has shown that this reaction cannot be relied on, for the violet colour may be obtained in this way when no bile-pigment is present.

I have myself repeatedly tried the same method, and I have seen it tried by others, but without any satisfactory result. Consequently I am not disposed to attach any practical value to it. Moreover, I think that there are theoretical considerations which have been overlooked, and which render it improbable that any considerable quantity of the biliary acids should be found in the urine in any case of jaundice. It seems to be forgotten that in health comparatively small proportions of those acids are discharged from the body. According to Bischoff not more than a quarter of the amount of biliary acids poured into the intestine by the liver passes away in the fæces, and even this has undergone important chemical changes. Evidently, therefore, the biliary acids subserve some further purpose in the economy, and it is probable that they are reabsorbed into the blood. But if this be the case we cannot expect to find them excreted by the kidneys as waste products in a case of jaundice.

It has already been mentioned that the question as to the presence of the biliary acids in the urine in jaundice is of consequence chiefly because it has been supposed to afford a means of distinguishing between two different forms of this affection. In some cases of jaundice it is found on post-mortem examination that there is an obstacle directly interfering with the flow of bile into the duodenum; in other cases no such obstacle can be detected. At first sight one can hardly avoid supposing that this must be a fundamental difference, and also that it should be sufficiently indicated by the state of the fæces, which one would hardly expect to be so entirely free from bile when the ducts are patent as when they are completely blocked up. We shall presently see, however, that there are many difficulties in the practical application of this distinction.

Simple Jaundice.—In a large proportion of cases of jaundice one can ascertain scarcely anything, whether by examining the patient or by asking him questions, beyond the facts that his skin and conjunctivæ are of a deep yellow colour, that the urine contains much bile-pigment, and that the fæcal evacuations are clay coloured. There is not, nor has there been, any pain or uneasiness in the region of the liver. Very often the patient says that he feels perfectly well, and would not know that anything was wrong with him but for seeing his yellow face in the glass. After a variable period the jaundice subsides. The first sign of improvement is generally that the motions return to their natural colour; the urine then soon ceases to contain bile-pigment, and a few days later the skin and conjunctivæ regain a healthy appearance. I have several times noticed that this favourable change has taken place about the twenty-first day; but in some cases it occurs earlier than this, and in other cases very much later. At no period of the complaint can one generally make out that the liver is enlarged, whether by palpation or by percussion; from beginning to end there is nothing to throw light on its cause. For a case of this kind no name seems so suitable as that of "simple jaundice."

Slow pulse.—There is, however, one symptom which is sometimes observed in this, but, so far as I know, in no other form of jaundice—I mean an extreme slowness or infrequency of the beats of the heart. The pulse occasionally falls to 50, 40, or even 20 in the minute. Now, a German observer, Röhrig, is said to have found in some experiments upon animals that the pulsations of the heart were much reduced in frequency by the injection of the salts of the biliary acids into the circulation.* It has therefore been supposed that the blood perhaps contains these acids in the cases of jaundice under consideration. But, as I have already stated, chemists have hitherto failed to discover these substances in the urine, whether in this or in other forms of jaundice; and, since they are readily diffusible, one cannot suppose that it is possible for them to accumulate in the circulatory fluid in sufficient quantity to affect the heart without being freely excreted by the kidneys. A very important question in regard to cases of jaundice in which the pulse is slow is whether the prognosis should be modified on that account. One is apt to be alarmed at it, and to dread the supervention of certain dangerous cerebral symptoms. But, in every case that I have seen, bile has before long again begun to pass into the intestines, and the patient has made a good recovery.

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* [Röhrig, 'Ü. d. Einfluss d. Galle auf. d. Herzthätigkeit,' 1863. See also a paper by Dr Legg ('Proc. Royal Soc.,' 1876) with references to Traube's observations.—Ed.]

Pathology.—As may be supposed from what has been stated in a previous paragraph, the complete absence of bile from the fæces in this form of jaundice affords a presumption that there is a mechanical obstacle to its flow. And the common theory is that simple jaundice depends upon *catarrh* of the larger bile-ducts. It is believed that their lining membrane is swollen, and that mucus is secreted, which obstructs the channel. I think, indeed that this condition has sometimes been demonstrated by dissection.

It has occasionally happened that an affection having the characters of simple jaundice has prevailed *epidemically*. Several instances of this were collected by Frerichs. Murchison mentions a remarkable outbreak of the same kind which occurred at Rotherham. In 1862 this town was visited by enteric fever, which proved very fatal. Early in the following year jaundice became epidemic. It is said that in February no fewer than one hundred and fifty persons were suffering from it, and there was this curious circumstance, that none of those who were attacked had passed through the fever. Murchison supposes that this form of jaundice is developed from a catarrh of the stomach and duodenum, itself induced by a chill, or by some other atmospheric influence. But some cases of simple jaundice are clearly traceable to causes which seem to exclude the possibility of their being due to a catarrhal affection of the bile-ducts. The complaint follows directly upon the shock of some mental emotion, which is generally of a depressing character.

Sir Thomas Watson mentions the case of a young medical friend of his who became jaundiced in consequence of his anxiety about an approaching examination before the Censors' Board of the College of Physicians; and he refers to another case in which an unmarried female, on its being accidentally disclosed that she had borne children, became in a very short time yellow. Almost every medical man has seen similar instances. Murchison places cases of this kind in an entirely different category from those which he attributes to catarrhal inflammation of the larger bile-ducts; but beyond the fact that the former are caused by mental emotion, while for the latter it is not possible to discover any cause, I believe that no difference can be found between them, either in their symptoms or in their course.

Diagnosis.—The characters which distinguish simple jaundice from other diseases in which jaundice occurs are chiefly negative; and one might expect that its diagnosis would be somewhat uncertain, since in cases of cirrhosis, of cancer, and even of gall-stones, jaundice is not very rarely the first symptom, preceding all the more especially characteristic symptoms of these several diseases. But whereas they seldom occur in young subjects and in those who have hitherto enjoyed good health, simple jaundice seems particularly apt to attack such individuals. Thus, it does not often happen that a mistake is made, and jaundice, attributed to catarrh of the bile-ducts, proves due to a more serious affection. Nevertheless, an absolutely favourable prognosis, without any reserve, ought not to be given in any case of what seems to be simple jaundice. We shall presently see that in many instances of acute atrophy of the liver jaundice precedes all the other symptoms by a period of several days, or even of some weeks. During this period the case cannot be distinguished from one of simple jaundice, and the most natural way of explaining the facts is to regard it as having usually at first been of this kind, and to look upon the fatal change in the structure of the liver as secondary and (as it were) accidental. Now, in acute yellow atrophy the ducts, as we shall see, are always found after death to be pervious and empty. But it is difficult to suppose that when a case of this kind begins as one of simple jaundice it is at first due to a catarrhal inflammation of the ducts, and that this afterwards subsides without leaving any indication of its having ever existed. As I have before mentioned, the fact that simple jaundice is frequently the direct result of mental emotion also presents a difficulty in the way of the acceptance of the current theory of its pathology. This question, however, is one which may be most conveniently discussed further on.

Acute Yellow Atrophy of the Liver.—I have just stated that in certain cases simple jaundice (as it had appeared to be), instead of subsiding favourably, becomes complicated by the supervention of cerebral symptoms, which rapidly destroy the patient. In these cases of *Icterus gravis* the liver presents very striking appearances, both to the naked eye and microscopically. And precisely the same appearances are observed in other cases, in which similar cerebral symptoms accompanied the jaundice from the first, arising suddenly in persons who had not before presented any indications of hepatic disorder. The affection is thus a very definite one, both clinically and pathologically, and its usual designation is "*acute yellow atrophy of the liver.*"

Anatomy.—The first thing that strikes one in making a post-mortem examination in a case of this kind is the diminution in the size and weight of the liver. The organ forms a thin flaccid mass, which lies at the back of the abdomen, hidden by the ribs and by the intestines, which last are commonly over-distended. Instead of weighing from fifty to sixty ounces—the usual weight of a liver at the period of life at which acute atrophy commonly occurs—it weighs perhaps thirty-two ounces, thirty ounces, or even as little as twenty-three ounces. When cut into, it looks as though it were softened; but the finger is found not to penetrate it more readily than the healthy organ. Although it is so flabby it is not really deficient in firmness. Its colour is greatly altered. Most of it has a bright orange-yellow tint; but some parts are dark red or purple. Sometimes roundish masses having this red or purple hue are scattered through the substance of the organ; sometimes one part, generally the left lobe, is almost entirely red, while the rest of the liver is mainly of a gamboge-yellow hue. To the naked eye it appears as though the red parts were less altered than the yellow, but the microscope shows that this is not the case. In both parts the hepatic cells have undergone destruction, and are replaced by a mass of granules and oil-globules; but in the red parts the destruction is complete, whereas in the yellow parts some of the secreting cells still remain visible, and towards the centres of the lobules may even retain their columnar arrangement. Often crystals of leucine and tyrosine are to be seen among the remnants of the hepatic tissue. The former substance presents the appearance of rounded, flat discs, generally marked with concentric rings; the latter occurs in bundles or globular masses of needle-shaped crystals. In some cases it has been thought that a pellucid nucleated material could be detected, supporting the detritus of the cells in acute atrophy. I believe that I observed this in two cases that have come under my own observation. Lastly, Waldeyer and Klebs have described, in the reddened parts, cells resembling those of the epithelial linings of the biliary ducts, arranged in regularly branching lines and tubes which seemed to have cæcal terminations.

The bile-ducts are found to be empty; their mucous membrane is unstained by bile-pigment. The gall-bladder is either empty or contains a few drachms of grey mucus or of a pale yellow or greenish fluid.

The kidneys can very generally be shown by the microscope to have undergone morbid changes. The epithelium of the lobules is granular and may be very fatty.

Symptoms.—In addition to the jaundice, these are mainly cerebral. Headache and intolerance of light are often first complained of. Before long the consciousness is more or less impaired. The patient now becomes very restless, screaming and tossing about from one part of the bed to another. Violent convulsions perhaps next occur. Ultimately a state of complete coma is developed; the pupils become widely dilated and insensible to light; the urine and fæces are passed involuntarily; the breathing is stertorous and terminates in the death of the patient, almost always within five days from the commencement of the characteristic symptoms. It is, indeed, said that acute yellow atrophy has sometimes destroyed life in less than twenty-four hours. According to Niemeyer the majority of cases end fatally on the second day, but this certainly does not accord with what I have seen of the disease.

Diagnosis.—The supervention of such alarming symptoms in a patient suffering from jaundice naturally excites the most anxious solicitude on the part of his medical attendant; and the clinical investigations of the last twenty years have led to the discovery of several objective signs, to be detected by careful examination, by which the suspicion of acute atrophy of the liver may be either confirmed or rejected. Thus, it is possible, by means of *percussion,* to trace from day to day the gradual diminution in size of the liver. From a normal measurement of four inches vertically in the right mammary line—reaching from the fifth intercostal space downwards to the costal margin—the hepatic dulness may be watched as it undergoes reduction to three inches, two inches, and one inch, until at last it disappears entirely. One must not, however, suppose that the diminution of dulness over the liver is necessarily due solely to the wasting of the organ. As we have seen, the liver in acute yellow atrophy is remarkably flaccid and falls backwards away from the ribs, so that the intestines, if distended, ride over it. Indeed, this source of fallacy is not confined to cases in which the liver is really affected with acute atrophy. In all forms of jaundice the bowels are apt to become inflated with gas, and the consequent enlargement of the abdomen may cause the right hypochondrium to become gradually more and more tympanitic, and the area of hepatic dulness to diminish from day to day. I have seen this lead to an error of diagnosis: the liver was believed to be in a state of acute yellow atrophy, where such was not the case.

Very important information is further afforded by the examination of the *urine.* This does not generally contain a very large quantity of bile-pigment. It does not look black when in bulk, nor in a thin layer has it so intense a saffron yellow colour as in some other forms of jaundice. It is said that Gmelin's test may give an imperfect reaction, or fail altogether to indicate the presence of the colouring matter of bile. The principal change in the urine is of a kind which at first sight would appear to have no relation to the disease in which it occurs. The urea and uric acid, and also the chlorides, sulphates, and earthy phosphates are greatly diminished in quantity or are altogether absent; and in their place are found two new substances—leucine and tyrosine—the same which have already been described as being present in the substance of the liver itself. There is generally no difficulty in detecting these substances. They sometimes form a distinct deposit when the urine is left to stand for a time, or, if this is not the case, they may be made evident by evaporating a few drops of it on a glass slide. In some cases, however, they cannot be discovered, at least without the adoption of a more complicated procedure. The urine is often albuminous.

With regard to the appearance of the *fæces* in acute yellow atrophy different writers have made different statements. Murchison says "the jaundice appears to be due to a poisoned condition of the blood, and consequently bile is still found in the stools." The remark has often been made that it is a good sign for the motions to be clay coloured in jaundice, there being then less danger of the supervention of cerebral symptoms. But it is at any rate certain that towards the end of a case of acute atrophy no bile enters the intestines, for after death the ducts and gall-bladder are found to contain an almost colourless mucus. Frerichs, indeed, says that in this form of jaundice the stools are dry and clay coloured, and I find more than one case recorded at Guy's Hospital in which such was the case. The question is not so easy of determination as might at first sight appear, on account of the great frequency of hæmorrhage into the intestines in this disease. It will be presently mentioned that in many forms of jaundice there is a tendency for the blood to escape from the vessels, and this is especially the case in acute yellow atrophy. Very frequently the patient vomits a dark fluid resembling coffee grounds, and containing altered blood. Petechiæ are often developed in the skin, and almost invariably, towards the end of the case, the evacuations have a dark brown or a tarry black colour, which is really the result of hæmorrhage. I am inclined to believe that when the stools have been supposed to contain bile in acute atrophy of the liver, their dark appearance has generally been due to altered blood. We must, however, remember that the disease does not affect the whole substance of the organ uniformly, but attacks some parts earlier than others. Thus, at its commencement bile very possibly continues to be poured into the intestines by those portions of the liver which have not yet become diseased.

When acute yellow atrophy occurs in a pregnant woman, *abortion* or miscarriage almost always precedes the patient's death.

The *cerebral symptoms* which show themselves in this disease are exactly like those of the "typhoid state" occurring in other febrile diseases. The patient has almost always a dry brown tongue, and his lips and teeth are encrusted with sordes. According to some writers, among whom is Niemeyer, the temperature of the body is raised considerably above the normal. But in several of the cases that have occurred at Guy's Hospital the temperature was normal. In one instance it was actually below the average four days before death, but it began to rise two days later, and while the patient was dying it was found to be 101·6°. Dr Duckworth noted the absence of pyrexia in three cases that were observed at St Bartholomew's Hospital. Frerichs says that in his cases the skin was usually cool, dry, and inactive, and he quotes Bright and Addison as having made particular mention of the same circumstance. The pulse is almost always accelerated, but in one of Dr Duckworth's cases it was on two days about 50. Towards the end it becomes very small and intermittent.

The prognosis of acute yellow atrophy is not always fatal. A few instances of recovery have been recorded.* Not long ago Dr Wilks had at Guy's Hospital a fatal case in which there was a distinct history of a previous attack that had been recovered from. The patient had become delirious and had such severe hæmatemesis that it was thought he would die in a few hours. However, he rallied and lived two months longer, at the end of which he again became delirious. Leucine and tyrosine were found in the urine. He died a fortnight later. The liver was found by Dr Moxon to weigh forty-seven ounces. The left lobe and the adjacent part of the right lobe were small and dark looking. The lobules in them were distinct, but scarcely any hepatic cells were to be seen. The rest of the right lobe formed a soft, yellow, rounded, projecting mass. The marked contrast appeared to justify the supposition that the left lobe had become atrophied at the time when the cerebral symptoms first arose.

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* See a case by the late Dr Frank Smith, of Sheffield; 'Path. Tr.,' 1877, p. 236.

Acute yellow atrophy of the liver is not a common disease, though by no means excessively rare. Murchison says that, although delirium and a brown tongue constituted a certain passport into the London Fever Hospital, only one case occurred among 30,000 patients admitted in a period of six years. In Guy's Hospital I find notes of the inspection of eight cases in twenty years.

The *ætiology* of the disease is exceedingly obscure. There are several different conditions, each of which has appeared in certain cases to be its exciting cause, and its occurrence seems to be favoured by other conditions, which must therefore be regarded as causes predisposing to it. Among the exciting causes mental emotions seem to take a foremost place. It has already been stated that fear or grief is a frequent cause of simple jaundice, and cases arising in this way may prove fatal by the supervention of cerebral symptoms. Again, more than one instance has been recorded in which it has followed directly upon a drunken debauch; and in a considerable number of cases it has set in during the secondary stage of constitutional syphilis. It might, indeed, be argued that one cannot in either of these conditions exclude the possibility that the jaundice was really due to the mental emotions arising out of the cause to which we attribute it. Persons affected with syphilis in particular often undergo an amount of mental torture which they carefully conceal, and which may well be supposed capable of affecting them very prejudicially. Pregnancy is another cause to which the disease is occasionally ascribed, and apparently with justice, for out of twenty-two female patients referred to by Frerichs one half were pregnant. But here again mental influences may possibly come into operation.

Even apart from pregnancy, acute yellow atrophy of the liver is more common in the female than in the male sex. It occurs principally at an early period of life. Five times out of six the patient is under thirty years of age. It is, however, very rare in infancy, although I have seen one instance of it in a child only two and a half years old. Graves has recorded the memorable case of two sisters who died of this disease in succession at an interval of eight months, while three months later a third sister was attacked with a jaundice which threatened to take the same course, but from which she recovered. It appears somewhat doubtful whether in these cases the disease is to be ascribed to an inherited predisposition, or rather to some obscure influence arising in the locality or house where the patients lived, for Dr Budd mentions that several sailors in the same vessel have been known to be attacked by acute atrophy of the liver.

Some of the earlier writers on acute yellow atrophy of the liver, having found after death that the larger bile-ducts were free from obstruction, conceived the idea that the minute channels which issue from the secreting lobules of the organ might have undergone compression in consequence of swelling of the cells forming the periphery of the lobules themselves. And they supposed that the jaundice was really due to reabsorption of bile secreted by the cells forming the centres of the lobules. Rokitansky even imagined that the breaking down of the hepatic cells was due to a solvent action excited by the retained bile. These views, however, can be controverted by evidence of great weight, and at the present day pathologists are pretty well agreed in believing acute atrophy of the liver to be *parenchymatous inflammation* (in Virchow's sense of the term), although one must admit that no precisely analogous disease can be found among the affections to which other organs are liable.

Some uncertainty still prevails with regard to the origin of the leucine and tyrosin, which, as we have seen, are excreted in the urine in this disease. Most authorities suppose that in acute atrophy of the liver the chemical changes which should be undergone by albuminous substances in the blood are incomplete, so that instead of urea and uric acid the new bodies in question are formed, and this view accords well with the fact that urea and uric acid are more or less completely wanting. But other writers, basing their opinion on the fact that the healthy liver during decomposition contains leucine and tyrosin, think that these substances are the direct products of the disintegration of the hepatic tissue.

Again, there is a doubt whether the granular and fatty changes in the epithelium of the renal tubules are the result of the disease of the liver, or whether both these conditions do not rather depend upon some common cause. The former view appears to me to be the correct one, for the morbid changes in the kidneys are after all often comparatively slight.

What is the cause of the cerebral symptoms which form so striking a feature in acute atrophy of the liver? Frerichs thought that they depended upon the presence of leucine and tyrosine in the blood, but experimenters have hitherto failed to verify this supposition. Rokitansky started the theory that these symptoms were really uræmic and dependent on the renal changes. But it seems to be an insuperable objection to this view that the character of the cerebral symptoms in acute atrophy of the liver is not the same as in uræmia.

The *diagnosis* of acute yellow atrophy of the liver is not in most cases difficult, if attention be paid to the various points to which I have referred in describing the disease. The affection which most closely resembles it is one produced by poisoning with phosphorus. Within the last few years it has been shown that the toxic effects of this substance are by no means limited to the vomiting and purging which immediately follow its ingestion. In a few hours these generally pass of, and often the patient appears to be perfectly well for three or four days, but at the end of this time jaundice sets in, followed by delirium and coma, and these symptoms ere long prove fatal. According to some observers, the liver is then found to be altered exactly in the same way as in cases of acute yellow atrophy. They therefore speak of phosphorus-poisoning as one of the causes of this disease, but I rather incline to the belief that the appearances are distinct from those seen in acute yellow atrophy. In some cases of poisoning by phosphorus at any rate, it is certain that the liver presents characters which are very different. It is larger than natural, of normal shape, and of a pale buff colour, mottled with numerous ecchymotic spots. Under the microscope the principal change is the presence of an immense quantity of fat, in large globules as well as in minute patches, within the hepatic cells; the walls of many of which are indistinct. One may therefore at first feel disposed to think that they have undergone destruction, but I have often found a similar difficulty in detecting the hepatic cells in other cases of fatty liver in which there was every reason to suppose that their walls were simply obscured by the fatty globules. Clinically also the effects of poisoning by phosphorus appear to be different from the symptoms of acute yellow atrophy. I am not aware that in the former affection leucine and tyrosine have been found in the urine. The liability to hæmorrhage, however, forms a prominent feature in both diseases. After poisoning by phosphorus the uriniferous tubules are loaded with highly-refracting granules, like those in the hepatic cells, and the fibres, both of the voluntary muscles and of the heart, are found to have undergone a granular fatty degeneration. Thus the morbid state produced by phosphorus appears to be an acute steatosis of the liver, kidneys, and muscles. In England such cases are rarely seen, but in Germany they are far from uncommon. Persons who wish to commit suicide there seem to use the heads of a bundle of lucifer-matches, just as among us they employ white arsenic for the same purpose. I have myself seen one example of poisoning by phosphorus which occurred in the practice of Dr Habershon at Guy's Hospital. In that case the temperature of the body was very low, at least for some hours before death, the thermometer standing in the axilla at 99·8°, and afterwards at 91·5°.

No *treatment* for acute yellow atrophy is known.

Febrile Jaundice.—If I am right in saying that in acute yellow atrophy the temperature of the body is seldom raised above the normal standard, this character may serve to distinguish that disease from some other affections of the liver no less fatal which are almost constantly attended with fever. These may be grouped together under the name of "febrile jaundice," a name which is more convenient in that the affections in question can rarely during life be distinguished from one another. In fact, if this or some similar expression be not employed, the cases in question must generally go without any designation beyond that of jaundice.

I am not now referring to the fact that jaundice may occur as a complication in some of the specific fevers. This is well known to be the case in "yellow fever" (vol. i, p. 258). Again, in "relapsing fever" jaundice is a frequent and not necessarily an unfavourable symptom (ibid., p. 161). It sometimes, though very seldom, occurs in typhus, and in this disease almost every patient who becomes jaundiced dies. In enteric fever and scarlatina jaundice is extremely rare. Another disease, attended with fever, in which jaundice may occur as a complication is inflammation of the base of the lung. This has been supposed to be due to an extension of inflammation through the diaphragm to the upper surface of the liver, and probably on this account it has been said to occur only in pneumonia of the right side, but at least one case of this kind has been observed at Guy's Hospital in which the left lung was the seat of the inflammation. Pyæmia, again, is often accompanied with jaundice; indeed a slight yellowness of the skin is one of the most frequent symptoms of pyæmia. Some years ago Dr Wilks investigated the question whether those cases of pyæmia in which abscesses occur in the liver are, or are not, particularly liable to be accompanied by jaundice, and he came to the conclusion that the local disease had nothing to do with the production of this symptom. I think, indeed, that in most of the febrile diseases of which jaundice may be a symptom, it is probably dependent upon changes in the blood rather than upon any morbid state of the liver.

Besides these general maladies there are certain diseases of the liver itself that are attended with fever, and at the same time more or less constantly give rise to jaundice. It may develop itself as an accidental symptom in cases of tropical abscess. But there is also a special form of suppuration in the liver in which jaundice frequently occurs.* This is the disease which has been termed *pylephlebitis*, or inflammation of the branches of the portal vein and of the connective tissue in which they are embedded. A very striking case of this kind came under my observation several years ago. A man, aged thirty-seven, was admitted into Guy's Hospital under the care of Dr Barlow (to whom I was then clinical clerk), exceedingly ill with jaundice, fever, and delirium; and he died in two days. He had been quite well a week before, except that he suffered from stricture of the rectum. The liver was very large, and its tissue was suppurating throughout. The branches of the portal vein were all distended with soft thrombi of a brownish colour. The main trunk of the vein contained a dirty-looking fluid. The inflammation of the liver was evidently due to the absorption of some unhealthy material by the veins ramifying in the coats of the rectum, which was extensively ulcerated. Not rarely suppuration of the portal canals throughout the liver is excited by the presence of gall-stones in the ducts. The possibility of this occurrence must never be forgotten in any case of biliary colic in which febrile symptoms show themselves, although (as will presently be stated) a high temperature may be present without any other evidence of inflammation having been set up.

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* [See, however, thirteen cases of "Multiple Small Abscesses of the Liver," recorded by Dr Carrington in the 'Guy's Hosp. Rep.' for 1882, p. 375, ten of which were due to Pylephlebitis.—Ed.]

Other causes of suppurative pylephlebitis which may be mentioned in addition to ulcerative affections of the stomach or bowels are suppuration of the spleen, suppuration of the mesenteric glands, and the penetration of one of the veins which go to form the portal trunk by foreign bodies; in a case quoted by Frerichs a fishbone had entered the inferior mesenteric vein.

Again, inflammation of the capsule that encloses a hydatid cyst of the liver not rarely sets up suppuration along the portal canals within the organ, which in this case, however, seems to depend upon inflammation of the branches of the bile-ducts rather than of those of the portal vein. The explanation of this lies in the fact that a suppurating hydatid very generally communicates directly with a branch of the bile-duct often of considerable size. Hence membranous portions of the hydatid, or of its capsule (detached by sloughing), often enter the bile-duct in these cases and may obstruct it. I remember a very interesting case of this kind which occurred in Guy's Hospital a few days later than the case of pylephlebitis that I mentioned just now. The liver was found after death to contain a suppurating hydatid cavity which held three pints of fluid. The bile-ducts throughout the organ were suppurating; the main canal was obstructed by a large piece of detached membrane rolled up into a cylinder.

Another rare cause of febrile jaundice is acute tuberculosis of the liver. An instance of this occurred to Murchison in a woman forty years of age. Another case was observed some years ago at Guy's Hospital. A man, aged thirty-seven, died with febrile symptoms of typhoid character. The history was imperfect, but he was stated to have had jaundice only five days. The jaundice was bright yellow. On post-mortem examination the liver was found to be full of tubercles, there being as many as fifty to the square inch. There was also pneumonia of the left lung.

From what has been stated in the previous paragraphs it appears that there are two main conditions which give rise to febrile jaundice, independently of those general diseases of which jaundice is an occasional complication. These are pylephlebitis and suppuration of a hydatid cyst with ulceration into a bile-duct. In both these diseases, besides the jaundice and elevation of temperature, there are generally pain and tenderness in the hepatic region, and the patient has repeated attacks of shivering. In attempting to diagnose between them one must be guided mainly by the clinical history. But it must be added that the distinction is of little practical importance, since cases of either kind very generally terminate fatally; only it is important not to overlook the presence of any suppurating cyst which may be within the reach of a trocar, as puncturing it early might, perhaps, give the patient a chance of recovery.

Jaundice from cirrhosis of the liver.—Writers generally state that jaundice is seldom caused by cirrhosis of the liver, and it is the fact that in the majority of cases this affection runs its whole course without interfering with the flow of bile through the common duct into the duodenum. But there is nevertheless a considerable minority in which jaundice is present, and not rarely it constitutes the chief symptom of the disease. I find that among one hundred and thirty cases, occurring in the post-mortem room of Guy's Hospital, in which the liver was found after death to be cirrhosed, jaundice existed in thirty-four, and in ten it was deep or intense. During this period, however, there were examined in the same place only some sixty other cases in which jaundice was a principal symptom. Thus, among the causes of jaundice cirrhosis of the liver is far from taking the insignificant place assigned to it.

The fact is that the frequency with which cirrhosis of the liver itself occurs is far in excess of that of the other serious diseases that give rise to jaundice; and, consequently, although jaundice is not a very common result of cirrhosis, cirrhosis is by no means an uncommon cause of jaundice.

Jaundice due to this disease has some peculiar characters. It is frequently gradual in its onset. It is often slight in degree. There is seldom, and perhaps never, a complete absence of bile from the fæces. It is unattended with pain, and so differs from most cases of jaundice due to gall-stones or cancer. On the other hand, it is very often associated with the other symptoms of cirrhosis which will hereafter be described, and particularly with ascites. I may take the opportunity of remarking that the concurrence of jaundice with ascites is met with in scarcely any other disease excepting cancer. Palpation and percussion afford very important indications in cases in which jaundice is caused by cirrhosis. As a rule, the liver is not contracted in these cases, being, on the contrary, increased in size. Thus, the area of dulness over the organ is greater than natural, and not infrequently the granular character of its surface can be distinctly made out during life. Those cases of cirrhosis which are attended with jaundice very generally end quickly in death; they often terminate by the supervention of cerebral symptoms, but I have met with an instance in which there was persistent jaundice for seven years, and in which the patient at last died (I may say accidentally) of hæmatemesis. In this instance the body never assumed the olive-green hue which is seen in other cases of long-standing jaundice, and I think that this change in the colour of the skin will be found never to occur in cases of jaundice from cirrhosis. As I have before remarked, it seems to take place only where there is complete obstruction of the bile passages, and this is absent in the disease now under consideration.

Jaundice from gall-stones.—Hitherto we have been dealing with varieties of jaundice in which there has either been no impediment whatever to the flow of bile into the duodenum, or in which the existence of such an impediment has been more or less uncertain. We must next pass on to consider certain forms of jaundice in which there is no such ambiguity, and in which a direct mechanical obstruction of the bile-duct is invariably present.

In the immense majority of cases, the jaundice now to be described is caused either by gall-stones or by cancer; and very frequently these two causes are combined. I must therefore deal with them together. It would indeed be very inconvenient in the midst of the present chapter for me to give a full account of the varieties and characters of biliary calculi. But I shall have to describe, further on, certain other effects to which these concretions give rise; and I will therefore defer for the present all details as to their nature.

A person who is attacked with jaundice from gall-stones generally experiences an agonising pain, which at once distinguishes this form of icterus from those that we have hitherto been considering. The pain begins unexpectedly and quite suddenly, often soon after a meal or after some muscular effort. It may be so excruciating that the patient is bent double by it and rolls upon the floor, uttering the most piercing cries and screams. Epileptiform convulsions may even be excited by it. After a little while its intensity becomes somewhat lessened, and it is then replaced by a constant dull aching which continues until the more acute pain returns. The seat to which these agonising sensations are principally referred is the right hypochondrium, but they generally also shoot into the right scapular region and back; and they often seem to spread over a large part of the abdomen. Another very marked symptom is shivering. The face is pale, the skin cool,* and the whole body often covered with a cold sweat. Vomiting is very frequent, and hiccough not uncommon. The pulse is much reduced in force and volume; sometimes it is slow, but more generally it is rapid and very weak. There is great exhaustion, the patient may swoon away, and it is said that fatal collapse may even set in. Jaundice is not one of the earliest symptoms of an "attack of gall-stones." Indeed, it is evident that, until the calculus has passed from the cystic into the common duct no jaundice will arise. Generally speaking, however, after a few hours, or at the longest a couple of days, the patient's urine begins to contain bile pigment, and jaundice shows itself a little later.

It is generally supposed that an attack of gall-stones (or "biliary colic," as it is often termed) is necessarily and invariably attended with pain. But a few years ago a case occurred in Guy's Hospital in which a man died in a surgical ward of hernia who had previously had jaundice, which (it is expressly said) was unattended with pain. The gall-bladder contained numerous gall-stones; and the common duct was dilated so that the finger could be introduced into it.

There are several different ways in which an attack of gall-stones may terminate. Most commonly the jaundice sooner or later subsides. Its duration is then very variable. Sometimes it passes off in three or four days, sometimes it lasts several months. Indeed, even the shorter period exceeds the limit within which an attack of gall-stones may sometimes run its course. It may terminate within twenty-four or thirty-six hours; but in that case it is unattended with jaundice, which, as we have seen, seldom appears until the pain has lasted for some time. A good example of protracted jaundice from this cause is given by Murchison. It is that of a man who was jaundiced continuously for more than six months. Even in this case, however, the pain was not constant, but repeatedly went away for a week at a time. At last the jaundice disappeared, and the man returned to work.

When an attack of jaundice from gall-stones subsides, it is usually because the calculus has passed into the duodenum. The next thing is for it to be voided in the fæcal evacuations, in which (if it be looked for) it may often be found without much difficulty. Formerly writers said that if water is added to a stool containing a gall-stone, the latter will rise to the surface, from being lighter than the liquid. But it is now known that this is a mistake. When first voided, gall-stones have really a higher specific gravity than water; it is only when they have been dried that they float in it. The best way to detect a gall-stone in the fæces is that recommended by Murchison, namely, to dilute the evacuation with water and to pass the whole of it through a sieve, or a piece of muslin. In some cases, however, after the subsidence of an attack, no gall-stone can be detected. Possibly it is retained for a time within the intestine, or it may have undergone disintegration, particularly if it was one of those friable calculi which consist almost entirely of biliary pigment; or, again, it may never have escaped into the duodenum, but may have slipped back into the cystic duct or the gall-bladder. The last alternative is the one that comes most naturally into one's mind when a patient has very numerous and transitory attacks of biliary colic in quick succession, and when yet no calculus can be found in the evacuations. One is tempted then to suppose that all the attacks are caused by a single gall-stone, slipping to and fro in the duct. On the other hand, it must not be forgotten that a very large number of calculi are sometimes present in the same gall-bladder, and that a great many have been found in succession in the fæces. Sir Thomas Watson relates the case of a patient who collected fifty-five calculi from his stools within a space of five weeks. The discovery of the concretion after an attack of jaundice is not only important as verifying the diagnosis, but it may also lend some help as regards prognosis, in reference to the question whether it is likely that the complaint will recur. If the gall-stone was alone in the gall-bladder, its form is rounded; but if it was one of several, it is very likely to show flat surfaces or facets where it touched the stones in contact with it.

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* Dr Duckworth has, however, seen several cases of biliary colic in which there has been pyrexia; in one of which the temperature rose nearly to 104°, and remained high for several days. Murchison also mentions that pyrexia is not uncommon ('Dis. of Liver,' p. 310).

I have already stated that according to writers on this subject an attack of gall-stones may be so severe as to prove directly fatal. But I am not sure whether any well-authenticated case can be adduced in which it has been shown by a post-mortem examination that death has actually been due to this cause, independently of any further morbid change in the ducts. What really sometimes, although rarely, occurs is that a gall-stone causes ulceration in the gall-bladder or in one of the ducts, which ulceration reaches the peritoneal surface, allowing bile to escape into the serous cavity, and setting up fatal peritonitis. Some years ago a case of this kind occurred in Guy's Hospital. A woman had for some few years had repeated attacks of jaundice. For four or five weeks she suffered continuously from this symptom and from pain in the abdomen, which became more severe, until she died. At the post-mortem examination acute peritonitis was discovered, which had been caused by the escape of bile through an ulcerated opening in the hepatic duct. The common bile-duct was obstructed by a gall-stone. Murchison relates the case of a lady who died in about a week from a second attack of jaundice, and in whom the fatal result was due to peritonitis set up by perforating ulcers in the fundus of the gall-bladder, themselves caused by gall-stones. In other cases the ulceration of the gall-bladder or biliary passages caused by gall-stones has set up a local pyæmia, attended with the formation of abscesses in the liver, and leading within two or three weeks to a fatal result. Cases of this kind must not be confounded with those already referred to in which diffused suppurative pylephlebitis is developed as a result of gall-stones.

Allusion has already been more than once made to the fact that biliary colic frequently occurs over and over again in the same individual, and a patient who has had this complaint once should therefore always be warned that he is likely to suffer from it again. Sometimes, but not always, the first attack is the most severe. When a concretion of some size has once passed through the common duct into the duodenum, it is, of course, easier for another calculus of the same size to perform the same journey. The cases in which biliary colic terminates rapidly within a few hours, and even without causing jaundice, are chiefly those in which several previous attacks of the same complaint have occurred. These instances of repeatedly recurring biliary colic are often very trying, both to the patient and to his medical attendant; but I believe that, in the majority of cases, the attacks sooner or later cease to return. At any rate, I know of several instances of this kind, even in persons advanced in years, who have afterwards enjoyed excellent health and have ultimately died of some other disease.

I have mentioned a case, recorded by Murchison, in which an attack of jaundice from gall-stones, after lasting six months, subsided, and the patient recovered. Sometimes, on the other hand, the jaundice persists until death. The termination of such a case may be due to some complication. In one instance that I have seen it resulted from erysipelas of the face; in another from the supervention, at the same time, of acute endocarditis and acute meningitis. In another case, again, the patient fell into a comatose state, and died a fortnight after the commencement of his last attack of jaundice; and a fourth case was probably similar, of which no history is preserved beyond the facts that it proved fatal a few days after admission to hospital, and that the body was well nourished, as in death from some acute disease.

During the twenty-one years from 1854 to 1874 inclusive, I cannot find a single case recorded in our post-mortem reports at Guy's Hospital in which death occurred with chronic exhaustion and wasting as the simple result of the jaundice due to impaction of a gall-stone in the common bile-duct. The twenty-five volumes of the 'Pathological Transactions,' again, contain only two such cases; and even these may be said to have proved fatal by complications. One is recorded by Murchison; the patient, who had for many years been subject to attacks of gall-stones, died after six months of jaundice, having suffered during the last three weeks from greatly increased pain and vomiting, hæmorrhages from mucous membranes, &c.; the common bile-duct was obstructed by a large cylindrical gall-stone, which was ulcerating into the bowel by the side of the orifice of the duct. The other case came under the observation of Dr Wale Hicks; the patient died seven months after the attack commenced, but the jaundice, instead of being persistent, gradually faded, and at last entirely disappeared, and the hepatic tissue was found to have broken down into granular matter and oil; moreover, the obstruction in this case was not complete.

But the way in which death is most frequently brought about in persons who have gall-stones is by the development of cancer about the gall-bladder or the bile-ducts. Within the period of twenty-one years already referred to, there have been at least twelve cases in which, gall-stones being present, there has been likewise malignant disease of these structures. In some instances the clinical history has pointed distinctly to the view that the jaundice was originally due to an ordinary attack of biliary colic, and that the development of the cancer was secondary; indeed, one case seems to admit of no other interpretation. A man, aged forty-five, died of jaundice which had lasted four months. Dr Moxon found that the gall-bladder was very large, containing hundreds of gall-stones; the common bile-duct at its commencement was greatly narrowed, and its walls were thickened by a cancerous growth; *below* the narrowed spot it contained three or four faceted gall-stones, just like those in the gall-bladder. This part of the duct also was dilated, and had evidently been accustomed to the passage of gall-stones before the cancer had begun to form. But, in the great majority of the cases in question, no gall-stone has been impacted in the duct at the seat of the cancer; the concretions have been found in the gall-bladder itself, which has often been contracted round them, and empty, or containing only a little purulent mucus. Thus it appears probable that if the malignant growth had not developed itself all the symptoms would have subsided and the health of the patient would have been restored. All observers, indeed, are not agreed that the cancer of the bile-ducts arises secondarily when it is found in association with gall-stones. It has been urged that gall-stones are often discovered in the bodies of those who have died from cancer of the breast or of other organs. This may be merely a coincidence, for both cancer and gall-stones are especially apt to occur in persons advanced in years. However, even if some deeper relation than at first sight appears probable should be proved to exist between the formation of biliary calculi and the development of cancer in the body generally, this would not do away with the clinical significance of the facts stated above: it would still remain true that when a patient who has had attacks of biliary colic dies of protracted jaundice the ducts are almost invariably found to be affected with cancer (v. *infra*, p. 284.).

Jaundice from cancer of the biliary passages.—Still more frequently, however, permanent jaundice is caused by cancerous disease alone, and independently of the presence of any biliary concretions. At Guy's Hospital cases of this kind have been just twice as numerous as those in which gall-stones existed. The exact locality and extent of the cancer have varied widely in different instances. In many of them its original seat appears to have been the head of the pancreas; in others its starting-point was the pylorus, or the first part of the duodenum. In some it seems to have commenced in the walls of the gall-bladder, or even in the liver itself, and to have passed downwards until it invaded the common bile-duct. In other instances, again, the cancer has affected the glands about the portal fissure, and then there has sometimes been primary cancer of some distant organ (such as the lower part of the small intestine). The extent of the cancerous disease, again, is very variable in these cases; there may be nothing more than a small nodule, no larger than a hazel-nut, involving the walls of the common duct. When this is the case, gall-stones are generally likewise present. Or all the parts in the portal fissure may be involved in an immense mass of cancer, which may extend to the peritoneum generally, or lead to the formation of large and numerous secondary nodules in the liver. In some of these cases, also, gall-stones have been found.

I believe that malignant disease of the biliary passages is almost always true carcinoma. Sometimes, indeed, the growth looks hard and dry, and yields little or no juice on scraping. But in one case, although the growth in the portal fissure looked as if it were composed of a fibroid material rather than like a true carcinoma, the liver contained large secondary nodules the character of which was unmistakable.

When there is a mass of cancer about the portal fissure, or in the lesser omentum, the portal vein is almost always pressed upon, and ascites follows. Thus, the association of ascites with jaundice is strongly suggestive of malignant growth outside the liver; indeed, with the exception of cirrhosis, this is the only disease in which these symptoms are often found together.

Other causes.—Permanent jaundice may be caused by other conditions besides impacted gall-stone and cancer. One of these is, in infants, a congenital *obliteration of the common bile-duct,* apparently resulting from intra-uterine perihepatitis; several instances of this have been recorded. The jaundice has appeared a few days after birth; it has been attended with hæmorrhage from the bowels and skin, and especially from the umbilicus. This last has sometimes been the cause of death, which in other cases has been due to progressive atrophy, attended with vomiting and diarrhœa; in two instances the child has lived as long as six months.*

Another very rare cause of permanent jaundice is *simple stricture* of the common duct, exactly resembling an ordinary stricture of the urethra. This condition is generally supposed to be the result of ulceration, itself set up originally by a gall-stone. Permanent jaundice may also be caused by an external *tumour,* of any kind, pressing upon the hepatic or common duct.

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* Jaundice in infants is not always due to this cause. It may arise from plugging of the common duct by inspissated bile, as in a case quoted by Murchison. But in many cases of supposed jaundice in newly-born children, the yellow tint is the result of changes of the blood in the over-congested skin, "the vivid redness of the newborn baby" (to use Murchison's expression) "fading, as bruises fade, through shades of yellow into the genuine flesh-colour."

Even tuberculous glands in the portal fissure have occasionally obstructed the flow of bile. Again, an abdominal aneurysm, particularly one affecting the hepatic artery, has sometimes proved to have been the cause of jaundice. Such cases have generally been attended with a severe paroxysmal pain, very like what would be produced by gall-stones.

Events of permanent jaundice.—Whatever its cause, jaundice with obstruction of the ducts, leads to a definite series of further changes. The gall-bladder and all the biliary passages become greatly dilated and distended, at first with bile, afterwards with a mucoid liquid, which may be of some shade of green or perfectly colourless. The gall-bladder may thus come to contain many ounces, or even (it is said) several pints, and it can be felt as a rounded or pear-shaped mass below the liver. Very often there are gall-stones in it as well as the fluid, and it is stated that their presence may sometimes be detected by palpation, and that they yield a peculiar crackling sensation, which has been compared to that produced by grasping a bag of hazel-nuts or rolling pebbles about in the mouth. Sometimes the gall-bladder in these cases suppurates and points externally. The result of this is the production of a fistulous opening, through which, after a time, green bile is discharged in quantities of from eight ounces to two pints daily. The jaundice may then subside; but this change is of no benefit to the patient, who becomes rapidly reduced in flesh and strength, and before long dies exhausted. The hepatic ducts, again, become dilated in cases of permanent jaundice; they may even become larger than the branches of portal vein with which they run, and may be visible on the surface of the liver as cylindrical tubes or sacculated pouches. The enlargement of the ducts appears to be the cause of the fact that the liver as a whole is larger than natural in the early stages of this form of jaundice. But after a time the organ begins to shrink, and at length it becomes considerably smaller than in health. Another change in the liver consists in its assuming a dark olive-green colour, which all writers describe as darker than that of other parts of the body. Doubtless this is due to the oxidation of the bile-pigment contained in the hepatic cells, converting it into biliverdin, exactly as in the skin. The connective tissue in the portal canals becomes thickened when the common bile-duct is obstructed. This has lately been dwelt upon by Dr Wickham Legg, who has shown that in the lower animals the operation of ligaturing the bile-duct is quickly followed by an overgrowth of connective tissue as great as in intense cirrhosis. My observations lead me to believe that a similar change occurs very frequently in cases of obstructive jaundice in man, although not to the same degree.

The liver-cells appear not to undergo any change beyond being somewhat reduced in size. In 1843 Dr Thomas Williams related a case in which jaundice was caused by malignant disease of the head of the pancreas, and in which almost all the cells of the liver were found to be broken down, fatty globules and granular matter being present in their place. But no one has since recorded a similar observation, except, indeed, when before death cerebral symptoms had been present of such a kind as to justify the opinion that the disease which caused the jaundice had ultimately become complicated with acute yellow atrophy. This occurred in a case which Murchison has placed on record in the 'Pathological Transactions' (xxii, 159).

Theory of jaundice.—We must now revert to a question of great importance, which I have already alluded to, namely, whether there are two distinct forms of jaundice, in the one of which the bile-pigment is secreted by the liver as usual, and afterwards reabsorbed into the blood; while in the other the secreting action of the liver is suppressed, so that any bile-pigment that may be present must have been formed by some other organ, or in the blood itself. We have seen that in certain cases of jaundice the ducts are mechanically obstructed, while in other cases they are patent. The question is whether the jaundice is essentially different in its origin in these two classes of cases. To use the phraseology of modern German writers, must we admit that there are two kinds of icterus—the one hepatogenous, the other hæmatogenous?

Now, one distinction between these two forms of jaundice has been supposed to be that bile acids are present in the urine in cases of obstructive jaundice, and are absent when the bile-ducts are free. I have already endeavoured to show that this distinction is untenable. So also in describing the different clinical varieties of jaundice, I have mentioned facts which show that the state of the fæces affords no such criterion. In acute yellow atrophy, in which the ducts are unobstructed, the motions are sometimes, if not always, free from bile; and, on the other hand, I have seen a case in which jaundice was due to obstruction of the common bile-duct by a cancerous growth, which, however, only partially occluded its channel, so that the fæces remained of their natural colour.

To some of my readers it may, perhaps, appear that the theory of a hæmatogenous jaundice is sufficiently refuted by the fact that it is inconsistent with the current physiological doctrine that the bile-pigment is in health formed by the liver, and does not pre-exist in the blood. But a glance at the evidence on which this doctrine is founded will show that the question cannot be thus summarily dismissed. This evidence (so far as it is independent of pathological observations) comes under two heads—1, in frogs the liver can be extirpated without jaundice being produced; and 2, no bile-pigment can be detected in the blood in health. Now (1) I hardly think that experiments on animals so low in the animal scale as frogs can be regarded as having much weight in the matter; and (2) it is quite conceivable that, even though the bile-pigment is formed in the blood, it may never accumulate in sufficient quantity to be detected by chemical tests, if we suppose that the liver, with its large and active circulation, is engaged in removing it from the circulating fluid as fast as it is produced. The remaining heads of evidence in regard to this question are pathological, and they have, I think, been misunderstood. Thus, Murchison lays stress on the fact that the gall-bladder and bile-ducts are sometimes found after death to contain only a grey mucus, although during life there had been no jaundice. This would be a striking argument if the liver were always diseased in such cases. But Dr Moxon found that of four instances of this kind, one only was a case of fatty liver accompanying phthisis, the others being cases of pyæmia or pneumonia. It is evident that these cases merely show that under certain circumstances the formation of bile-pigment may be arrested. They have no bearing on the question whether the bile-pigment is formed by the liver or elsewhere.

Murchison, although a firm believer in the doctrine that the bile-pigment is formed by the liver, nevertheless does not adopt the opinion that the presence or absence of obstruction of the ducts makes any important difference in the way in which jaundice is produced. He points out that the osmotic currents between the blood and the contents of the biliary passages and intestines are extraordinarily active, and that even in health much of the bile which is secreted by the liver is probably reabsorbed into the blood. He argues that we have only to suppose that this reabsorption is excessive, or that the reabsorbed pigment fails to be properly got rid of, and we have at once an explanation of the occurrence of jaundice with patency of the ducts. But, as Dr Moxon has pointed out, this theory, that jaundice is in all cases due to reabsorption, is entirely inconsistent with the fact that in jaundice the biliary passages are almost always found to contain, not bile, but an almost colourless mucus. This is the case, not only in acute yellow atrophy of the liver, but also when the ducts are permanently obstructed by cancerous growths, gall-stones, &c. As Dr Moxon remarks, the contrast is at first sight very astonishing between the deep yellow fluid found in distant serous cavities in such cases and the clear liquid which is present in the ducts of the liver itself, and which is sometimes so completely devoid of bile-pigment as to yield no reaction with Gmelin's test. The absence of bile from the biliary passages is, however, easily explained. Physiologists have shown that the secretion of bile takes place under very low blood-pressure. Hence, when the common duct is obstructed, the entrance of bile into the biliary passages is probably arrested almost instantaneously. But the liquid poured out by the walls of these passages themselves and by the gall-bladder can undoubtedly continue to be formed under a much higher resistance. Consequently it soon displaces the last trace of bile; and like all mucous fluids, it is itself unstained by bile-pigment, even when jaundice is present.

This explanation evidently assumes that a colourless mucus will be found in the bile-ducts in cases of obstructive jaundice only when the obstruction is complete. And on searching the records of post-mortem examinations at Guy's Hospital for the last twenty years I fail to find any exception either to this rule or to that from which it is derived. On the one hand, I find no case of long-continued complete obstruction in which the ducts contained a liquid highly charged with bile-pigment. On the other hand, I find no instance of partial obstruction in which they contained a colourless mucus.

Certain writers have endeavoured to account for the presence of a fluid unstained with bile in the biliary passages in cases of jaundice, by supposing that the smaller ducts are plugged with inspissated bile, which thus cuts them off from the canals into which they should open. But it is surely inconceivable that all the smaller ducts throughout the organ should at the same time be obstructed in this way. For there would be no alternative but to suppose that in all cases of complete obstructive jaundice the bile-ducts up to the very point where their radicles meet the walls of the hepatic cells are plugged with a non-diffusible mucous fluid. This evidently brings obstructive jaundice into very close proximity with that form of jaundice in which there is no obstruction, and I think it can hardly be doubted that they both arise in essentially the same way.

The question as to the origin of jaundice is now reduced within very narrow limits. I fear that I must still leave it unanswered. Is the bile pigment first formed by the hepatic cells from materials which the blood brings to them, and is it immediately afterwards given up to the blood again? Or is it formed elsewhere in the body, so that the healthy liver-cells secrete without producing it and have their functions abolished from the time that jaundice appears?

In favour of the latter view is the fact that in protracted jaundice the liver-cells contain bile-pigment which has stagnated in them so as to have become green by oxidation. Moreover, it is difficult to see why jaundice should ever occur without obstruction of the ducts if the former view is correct. I could much more easily believe that in acute yellow atrophy the function of the liver is completely arrested than that the liver-cells pour back the bile-pigment into the blood as soon as it is formed by them, the ducts being perfectly patent. On the other hand, acute yellow atrophy itself affords one argument of great power in favour of the view that the bile-pigment is formed by the liver. This lies in the fact, pointed out by Murchison, that as the destruction of the liver-cells progresses, the amount of pigment that is excreted by the kidneys diminishes.

On the whole, I incline to the belief that in jaundice the bile-pigment is really first secreted and then reabsorbed.

Other effects of jaundice.—Certain minor phenomena, which sometimes accompany jaundice, now demand a brief notice. Among these is a deranged state of the functions of the alimentary canal. In 1832, Bright recorded some cases of jaundice in which the alvine evacuations contained a substance like fat, which either passed separately from the bowels or soon divided itself from the general mass, and lay upon the surface; "sometimes forming a thick crust, particularly about the edges of the vessel, if the fæces were of a semi-fluid consistence; sometimes floating like globules of tallow which had been melted and become cold, and sometimes assuming the form of a thin, fatty pellicle over the whole, or over the fluid parts in which the more solid figured fæces were deposited." This state of the fæces was sometimes so marked as to have been noticed by the patient before Bright saw the case. The oily matter had generally a slight yellow tinge and a most disgustingly fœtid odour. Bright was himself disposed to regard this peculiar condition of the evacuations as due to disease of the head of the pancreas and of the duodenum, and he seems to have thought that the presence of jaundice in his cases was accidental. Later writers also have generally attributed the symptom under consideration to obstruction of the pancreatic secretion, supposing that it prevented the fatty matters taken in the food from being digested and absorbed. But this appears to be too narrow a view of the subject, for Bidder and Schmidt found that in dogs, in which the bile-duct was ligatured, the amount of fat that could be absorbed from the intestines was reduced to less than one half, and sometimes even to as little as one fifth or one seventh of the quantity that the animal could digest before. These experiments suggest a doubt whether the presence of fat in the fæces in Bright's cases was not caused by the obstruction of the common bile-duct rather than of the duct of the pancreas. But the symptom is in fact one which is seldom present. Bidder and Schmidt's observations also established the fact that in dogs in which a biliary fistula had been formed, the quantity of food necessary to maintain the nutrition of the body is very much larger than before. This corresponds well with the circumstance that in all protracted cases of jaundice the patient becomes exceedingly thin and emaciated; although few cases of jaundice are entirely free from other conditions which might also account for the wasting.

Another result of the absence of bile from the bowels in jaundice is that their contents undergo putrefactive changes, the occurrence of which is prevented under normal conditions by the powerful antiseptic properties of bile. Hence the evacuations often have a very fœtid odour, and gases are generated which cause tympanitic distension of the abdomen. The contents of the intestines may probably in this way acquire irritant properties and so set up diarrhœa. But, on the other hand, obstinate constipation is very often present in jaundice, and this is commonly accounted for by the hypothesis that the bile is the "natural purgative" or stimulant to the peristaltic action of the bowels.

Again, jaundice is sometimes attended with great itchiness of the cutaneous surface. Now, in certain persons papules develop themselves whenever the skin is scratched at all vigorously or for any length of time. Hence when they are jaundiced they often present an eruption of pimples, the summits of which become quickly destroyed by the finger-nails. I have known this rash to be mistaken for scabies. According to Graves, urticaria may develop itself under the same circumstances. This accomplished physician also pointed out the fact that itching of the skin sometimes precedes jaundice by a considerable interval; in one of his cases this was a period of ten days, in another of two months. I remember that the late Dr

Addison used to maintain the same fact. He even said that he had once suggested beforehand the possibility that an attack of jaundice might be impending when a patient complained of itching, for which no explanation could be found, and that his prediction had been justified by the result.

Xanthelasma.—This will be the best place to mention another cutaneous affection which also accompanies jaundice, but only jaundice of very long standing. It was originally described by Addison and Gull under the name of Vitiligoidea, but it is now generally designated *Xanthelasma*, the latter name (from ξανθὸς and ἔλασμα = "yellow lamina") having been invented for it by Sir Erasmus Wilson.* Those who have access to the volumes of the 'Guy's Hospital Reports' or to the 'Pathological Transactions' † will obtain a better idea of this curious affection from the illustrations of it that have been published than from any description that I can give here.

It shows itself in two or even three distinct forms. There are in the first place flat cream-coloured patches (Vitiligoidea-plana of Addison and Gull) which are perfectly soft and cannot be detected by the finger, although they look raised and have defined margins. The first indication of the development of xanthelasma is always the appearance of such a yellow patch in one of the upper eyelids, just above the internal canthus. Afterwards other patches come out in the same neighbourhood, and these may ultimately coalesce so as to form a broad ring surrounding nearly the whole of the orbital regions. Similar flat patches also show themselves elsewhere; on the surface of the body, on the backs of the hands, in the scrotum, and also on the palms and soles, where they either present a peculiar dotted appearance or form long streaks following the creases of the skin. This plane variety of xanthelasma may also affect mucous membranes as well as the skin. Thus I have shown that it occurs in the gums and palate and in the larynx and trachea. Dr Legg has seen it on the side of the tongue, and in two cases I have known the lining of the bile-ducts to be covered with it. The next form of xanthelasma consists of raised solid nodules or even tumours (Vitiligoidea-tuberosa). These, I believe, are always later making their appearance than the flat patches. They occur on the ears and on the limbs (especially on the extensor surfaces) in a form which reminds one of the larger papular syphilides; they constitute large aggregated tubera over the olecranon; on the knuckles they form swellings very like those that may result from gout, and some of them are sometimes seated not in the skin itself, but in the adjacent tendons of the extensor muscles of the fingers.

Xanthelasma is clinically important not only because it is interesting to the physician, but also because it is often attended with much suffering to the patient. The parts affected with it are sometimes exceedingly tender. One patient who came under my observation was unable to stand or even to sit with any comfort, on account of the pain produced by the slightest pressure on the xanthelasmic patches on the feet and buttocks respectively, and for a similar reason she could not use her needle. In this case the affection became much less marked under medical treatment, most of the raised tubera disappeared, and the pains were in great part removed.

Microscopical examination shows that xanthelasma is essentially due to a fatty degeneration of the structures which are affected by it, the yellow colour depending upon the presence of innumerable fatty granules in the tissue. In the nodules there is also present a dense fibrous tissue, and even in the "plane" variety a few ill-formed cells have been detected which are apparently the result of a slight chronic inflammation of the tissue. The minute structure of a xanthelasmic patch is thus identical with that of an atheromatous patch in an artery, and it is interesting to note that in one case which I have seen the pulmonary artery and the lining of the left auricle was atheromatous as well as the aorta. This analogy appears to afford a clue to the pathological nature of xanthelasma, which I suppose to be a fatty degeneration consequent on a slight chronic inflammation of the tissue affected, this inflammation being itself caused by irritation from the continued presence of bile-pigment in its substance.

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* Xanthoma (yellow tumour) was suggested by Dr Frank Smith ('Journ. of Cut. Med.') 1869, and has been generally adopted in Germany.

† [See 'Guy's Hosp. Rep.' for 1851 (Addison and Gull's original paper), 2nd series, vol. vii, p. 267 (plates); ibid., 1866, p. 277 (plates); ibid. 1877, p. 97, with thirty-eight tabulated cases; 'Path. Trans.,' 1866, p. 277 (plates); ibid., 1868, p. 436; ibid., 1882, p. 376, with thirty-six tabulated cases of multiple xanthelasma (plate xx, 2).—Ed.]

It must not be forgotten that there is also a local form of xanthelasma which is confined to the eyelids, and which Mr Hutchinson has shown to occur chiefly in those who have suffered greatly from sick headaches (see vol. i, p. 672). There are also one or two cases of diffused or multiple xanthelasma on record in which jaundice has been absent.* The relation of xanthelasma towards the severe forms of jaundice that have been described is, I believe, that it arises whenever a jaundice lasts for a considerable time. In most of the cases that I have seen, it did not make its appearance until the patient had been jaundiced for a year or a year and a half, but in a case of Dr Pye-Smith's it began within six months after the jaundice, or perhaps even earlier. Hitherto it has been observed much more frequently in women than in men. From what has been already said it follows that xanthelasma is but very little likely to occur in those forms of jaundice which have directly fatal tendencies. Hence, in a large proportion of the cases in which it is developed, the obstruction of the bile-ducts is incomplete, and the skin consequently remains of a bright yellow colour, however long the jaundice may last. One of my patients had been jaundiced continuously for seven years before her death, but her evacuations all along contained bile, and she never acquired the olive-green colour which is seen in a protracted case of this kind whenever the common bile-duct is completely obstructed. On post-mortem examination cirrhosis of the liver was found to be the only cause of the jaundice.

Treatment of jaundice.—We have now to consider what remedial measures are applicable to jaundice, to the various diseases of which it is a principal symptom, and to the secondary symptoms which in their turn depend upon it.

Simple jaundice is so uncertain in its course that it might well appear a hopeless task to determine whether remedies are capable of abridging its duration. But in many cases the disease subsides so quickly after the commencement of a certain plan of treatment that it is difficult to resist the conclusion that these are possessed of very decided curative powers. On the Continent the most efficacious remedy is believed to be the administration of certain mineral waters: those of Vichy, Ems, Kissingen, Marienbad, and Carlsbad. Of these Vichy is the one most strongly recommended by French physicians, while German writers speak most highly of Carlsbad, which Niemeyer (for instance) describes as brilliantly successful in cases of simple jaundice. And since all these springs contain a considerable quantity of the salts of soda (especially the sulphate and carbonate) it is interesting to find that the same salts are believed in this country to be very useful in the treatment of simple jaundice. It is true that we seldom give the salts of soda alone, but rather combine them with remedies such as taraxacum and rhubarb, which are either thought to exert a specific action on the liver, or are regarded as useful by regulating the bowels. That in this way it is possible to bring an attack of jaundice to an end I feel confident, and I think that the following cases which came under my observation go far to prove it. A man, aged fifty-nine, came to me on January 9th, 1874, suffering from jaundice, which was not very deep but had already been of two months' duration. There was some tenderness and fulness over the liver. I ordered him to take half a drachm of spiritus ammoniæ aromaticus in a mixture of rhubarb, soda, and calumba. He came again on the 16th and said that for five days his motions had continued to be clay coloured, but that on the 14th they began to contain bile and that they were now quite dark coloured. On testing his urine I found that it contained very much less bile pigment than before. His jaundice was much diminished, and in the course of another week it entirely disappeared. In the next case there was some doubt as to the exact cause of the jaundice, but this does not affect the question of the value of the treatment. A bargeman came to me on February 23rd, 1872. Ten months before he had become suddenly jaundiced without pain. A month afterwards he had been seized with excruciating pain over the liver, lasting some hours, and he had since had three similar attacks. He had been under treatment both in Guy's Hospital and at King's College, but without result. He remained jaundiced the whole time. Having ascertained the treatment that had been previously employed in Guy's, I ordered him to take ten grains of carbonate of soda, with a scruple of extract of taraxacum and (as he had some dyspeptic symptoms) half a drachm of tincture of hop three times a day, and a grain of opium at night. On March 1st he came to me again and assured me that he was very much better, having lost the pain and sickness. On March 4th he noticed that his motions resumed their natural appearance, and before long he was well. I do not suppose that all cases of simple jaundice will subside equally rapidly under such treatment, but I think that it certainly deserves a fair trial. Another remedy which is often prescribed for this complaint is the dilute nitro-hydrochloric acid. I have seen simple jaundice quickly pass off in patients who have been taking this remedy also.

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* [Five have now been recorded in adults against twenty-three with jaundice, but some of the five are doubtful. Of eight cases of congenital or infantile xanthelasma multiplex, not one was associated with jaundice; and in these the eyelids were not affected, as they always are in adults. See 'Path. Trans.,' vol. xxxiii, p. 383.—Ed.]

With regard to the general management of the disease, the patient should not be kept in bed, nor even (in fine weather) within doors. He should have cheerful society and be allowed to take moderate exercise, and to have a nutritious diet, from which, however, fat, pastry, malt liquors, and the like should be carefully excluded.

When the cause of the jaundice has been removed, the fading of the yellow colour of the skin may, according to Murchison, be facilitated by warm baths, by giving the patient diuretics and diaphoretics, and by the administration of benzoic acid in four-grain doses three times a day.

In *acute yellow atrophy* of the liver no remedies have yet been shown to be generally efficacious. The cases that I have seen have not had any active treatment, it having rather been taken for granted that they must necessarily terminate fatally. However, I have already referred to one case which shows that there are exceptions to this rule, and, since the disease seems not to attack the liver as a whole, but generally to spread through the organ from the left lobe, there appears to be no reason why it may not be opposed by medicines. Dr Budd used to recommend a mixture containing a drachm of the sulphate and fifteen grains of the carbonate of magnesia with half a drachm of the spiritus ammoniæ aromaticus three times daily. This advice seems to have been founded upon the brilliant results which certain Irish physicians formerly obtained from purging in such cases. As striking examples, I may take the cases recorded in the year 1834 by Dr Griffin, of Limerick. Four children of the same parents were attacked within a few weeks by jaundice, with cerebral symptoms. Two of them died, but two recovered after having been in a state of almost complete coma. The treatment consisted of bleeding from the temporal artery, cold to the head, blisters to the neck, and above all, active purging, castor-oil being administered every fourth hour. These cases certainly indicate the expediency of a further trial of a similar practice.

With regard to the treatment of the various disease which may give rise to *febrile jaundice* there is but little to be said; they are perhaps even more invariably fatal than acute atrophy itself. The administration of quinine and stimulants may doubtless somewhat lower the pyrexia, and if the issue should be at all doubtful, may perhaps incline the balance in the patient's favour.

In an attack of *gall-stones*, or "biliary colic," the patient should first be placed in a hot bath. Fomentations and poultices are then to be applied to the abdomen. And if there be much tenderness on pressure in the right hypochondrium, a few leeches may often be used with great relief to this symptom, according to the testimony of Murchison and other writers. But these measures will not suffice for the relief of the agonising pain without the administration of opium or morphia in full doses frequently repeated. In a patient previously in good health two grains of opium are not too much to begin with, followed by a grain every two or three hours, until ease or sleep is obtained, it being of course understood that the case is carefully watched. Very often the stomach is too irritable to retain the anodyne, and then the subcutaneous injection of a quarter of a grain of morphia may be resorted to with signal advantage. Another antispasmodic, of which Murchison speaks highly, is the extract of belladonna, which he gives for this purpose in half-grain doses. The inhalation of chloroform or ether has sometimes proved very effectual. Another plan, which was highly recommended by Dr Prout, is the administration of large draughts of hot water, containing one or two drachms of the bicarbonate of soda to the pint. Dr Prout speaks of this as acting like a fomentation to the seat of pain; and even when the stomach rejected the first portion of the fluid, he used to persevere, believing that it diminished the severity of the retching. If, however, the vomiting be very violent, it should be checked by effervescing draughts, dilute hydrocyanic acid and the like. Many of the older physicians, and even Bright, prescribed antimony in the treatment of biliary colic with the hope of relaxing spasm, and so facilitating the expulsion of the calculus, but this remedy is now justly discarded on account of its tendency to aggravate the irritability of the stomach.*

When *permanent* jaundice has once declared itself, and the obstruction of the common duct is complete and irremediable the treatment should be carefully adjusted to the condition of the patient. It is no longer advisable to prescribe carbonate of soda, or dilute nitro-hydrochloric acid, or taraxacum. The more faith we have in the efficacy of these remedies in simple jaundice, the more disposed we shall be to fear that they may do harm now. Regulation of the diet is perhaps the most important part of the treatment. It has been shown experimentally that dogs with artificial biliary fistula may live for years provided they are supplied with and will take a sufficiently large quantity of food. It is true that in those animals the escape of bile through the fistula causes a drain which is wanting in jaundice in the human subject, but the experiments to which I have referred at least suggest the conclusion that a large supply of nutriment should if possible be maintained. At the same time its quality should be carefully attended to. I have already alluded to the experiments of Bidder and Schmidt, which showed that in dogs whose common duct had been tied the daily quantity of fat that could be absorbed from the food was greatly diminished. Evidently, therefore, oleaginous and fatty articles of diet should be taken very sparingly, if at all, by persons with permanent jaundice. Something may, indeed, be done to counteract the absence of bile in the intestines by the administration of the purified bile of the ox or pig. Murchison recommends that this should be given in doses of from three to six grains, about two hours after meals, in capsules or pills coated with a solution of tolu in ether, so that they may pass through the stomach unaltered. The pancreatic emulsion may also be fairly expected to do good in such cases. Oxgall also has the advantage of taking up the antiseptic function of the natural bile. With this object, as well as that of relieving flatulence, creosote, turpentine, vegetable charcoal, &c., may likewise be prescribed with benefit. Occasional laxatives are generally required, and the milder ones should be preferred.

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* [The treatment of gall-stones and of a suppurating gall-bladder by operation (cholecystotomy) has been followed in recent times by some remarkable successes: see Dr Marion Sims' case, 'Brit. Med. Journ.,' June 8th, 1878 (p. 811); Mr Lawson Tait's, 'Med.-Chir. Trans.,' 1880. References to earlier proposals of the operation, and isolated cases of its execution, will be found in the 'London Med. Rec.,' April 15th, 1881, p. 153.—Ed.]

The *itching* caused by jaundice is sometimes so troublesome as to require special treatment. Warm baths have sometimes proved serviceable, and, according to Niemeyer, especially alkaline baths. The use of the flesh-brush is recommended by Murchison, and the internal administration of the bicarbonate of potass. He mentions that the last-mentioned remedy did some good in one case in which opium and morphia had failed. Very often, however, this symptom is one which baffles all efforts to relieve it.

Gall-stones.—In speaking of the jaundice as caused by gall-stones, I found it convenient to postpone for the time a description of the nature of these concretions, and I must now revert to them for the purpose of repairing the omission, and of drawing attention to some other effects which may be caused by them.

Gall-stones are principally of two kinds. Some of them consist almost entirely of bile-pigment, the amount of cholesterine in them being very small; others are made up mainly of cholesterine with a proportionately less quantity of pigment, which last may indeed be altogether absent from the more superficial parts of the concretion. The former kind are very constant in appearance. They are small, of a dark green or almost black colour, irregular in outline, and often nodulated, and so soft that on pressure they break down into a rough gritty substance. The latter kind are hard and smooth on the surface; they split with a semi-crystalline fracture, displaying lines radiating from their centre, and the glistening aspect of cholesterine. In size and colour they are very variable. Some are three and a half to four inches in circumference. A gall-stone of this size is generally single and fills the whole gall-bladder, within which it appears to have been moulded, so that it has one rounded end answering to the fundus, and another tapering and truncated, which corresponds with the entrance of the cystic duct. Others are quite small, of the size of marbles, peas, or scarcely larger than pins' heads. Several of them are generally found in the same bladder, and sometimes an enormous number—as many as 1300, or even more. Their surface is commonly, but not always, white or stone coloured, but their interior has generally a more or less deep yellow or brown hue from bile-pigment; and this is often arranged in concentric layers. I believe that the centre of every gall-stone consists of biliary pigment.

The liability to the formation of gall-stones in the body increases very greatly as persons advance in years. In the large majority of cases, patients who have them are over fifty years of age, and many are sixty or even seventy years old. Sometimes, however, these concretions are found in those who are not older than twenty-five or thirty; and Cruveilhier and others have recorded their presence in infants and young children.

Women are more liable to them than men, in the proportion, according to Hein, of three to two. Perhaps this is due to the fact that females over the age of forty are particularly apt to gain flesh and to lead very sedentary lives, for such conditions are believed to be concerned in the production of gall-stones.

The immediate cause of their formation is supposed to be due to a decomposition of the bile. This leads to the deposition of the biliary colouring matter, and so a nucleus is formed, upon which cholesterine is slowly deposited.

Gall-stones are often found in the dead bodies of those who during life had not complained of any symptoms that could have suggested a suspicion of their presence. The gall-bladder is sometimes closely contracted over them, and (as its duct is generally obstructed by one of the concretions) it may contain beside only a very small quantity of mucus or pus. In such cases no clinical interest appears to attach to their presence. But sometimes when the cyst appears to be thus blocked, the walls of the gall-bladder go on pouring out mucus. It may then become much enlarged and form a tumour in the abdomen below the liver, the real nature of which is not always easy of determination. It may, for instance, be mistaken for a pendulous hydatid cyst.

The inflammation of the gall-bladder set up by gall-stones may also spread to adjacent parts, and so give rise to a variety of symptoms. Thus, the distended gall-bladder occasionally becomes adherent to the abdominal parietes; an abscess may then be developed which points externally. And when it breaks or is opened by a surgeon, gall-stones are discharged with the pus. The site of the external opening is by no means always directly over the gall-bladder; it may be at the umbilicus, or even in the left side of the abdomen; nay, a case has been recorded in which two biliary calculi made their way into the connective tissue of the vagina. It is important to note that in cases of this kind there are (or may be) no symptoms directly suggestive of the presence of gall-stones until they are found in the discharge. The common bile-duct is often quite free, while the cystic duct is completely closed; and thus, neither is there any jaundice nor does any bile enter the gall-bladder and mix with the pus. Hence, the abscess is often supposed to be seated in the abdominal walls, or, again, in the substance of the liver, and months or even years may pass before the real nature of the case declares itself. In the meantime the patient has a fistulous opening in the side, which, however, need not prevent the enjoyment of good health; and when all the gall-stones have come away, it may, at length, heal up.

In other instances the gall-stone escapes by ulceration, not through the parietes of the abdomen, but into some part of the intestine. It may be voided per rectum; and since a concretion which takes this course is often very large its passage through the anal orifice may be attended with severe pain and violent straining, the cause of which cannot be explained until the gall-stone is discovered. Probably, when a large stone thus makes its way out of the body, it had passed from the gall-bladder directly into the hepatic flexure of the colon. But much more often, it is with the duodenum that an ulcerated gall-bladder communicates. And, as I have already mentioned, the gall-stone may then fail to pass through the small intestine, and becomes impacted in the jejunum or the ileum, setting up fatal obstruction of the bowels. The least frequent event of all is for the gall-stone to be vomited. Murchison thinks that in such cases the gall-bladder opens directly into the stomach by ulceration.

CIRRHOSIS OF THE LIVER AND ASCITES

CIRRHOSIS—*Anatomy—Ætiology—Clinical symptoms and course—Hypertrophic cirrhosis.*

ASCITES—*Diagnosis—Ovarian dropsy—Perihepatitis—Atrophy—Gummata—Carcinoma—Pressure on, and obstruction of, the portal vein—Prognosis—Treatment—Paracentesis.*

In the last two chapters we have been discussing diseases which we believe to be caused more or less directly by disorder of the hepatic functions, but in most of which the liver itself cannot be said to present any definite pathological changes. We have now, on the other hand, to deal with a morbid condition, in which the structure of the liver is very greatly altered, but in which there is often very great difficulty in detecting any impairment of function. The condition in question is that known as cirrhosis, or granular disease of the liver; or more familiarly as gin-drinker's or hobnailed liver.

Anatomy.—A liver affected with cirrhosis presents very remarkable characters. Instead of being red it is pale, and mottled with grey and yellow tints. It is exceedingly tough and hard, so that one may be unable to crush it by the pressure of the finger; its tissue may even creak when cut with a knife. Its surface is not smooth and even as in health, but presents numberless round elevations, of all sizes, from the minutest granules up to the size of peas, or even larger. Similar bodies are closely packed throughout its substance. They are of a bright yellow colour, and on this account Laennec invented the name of cirrhosis for the disease (*κιῤῥός*, yellow). At this time, indeed, they were regarded as morbid products, and Morgagni and others spoke of them as tubercles. The microscope, however, shows that they consist of liver substance, which may be hardly distinguishable from the tissue of the healthy organ. The material which is really morbid is that which lies around and between the yellow bodies. This is a greyish, somewhat translucent substance, which under the microscope is found to consist of cellular elements and of fibrous tissue, in different proportions in different cases. Sometimes, but very seldom, it is made up entirely of cells, constituting what is termed embryonic tissue; this is in the earliest stage of the disease, when death rarely occurs. In the great majority of instances it consists of well-developed fibrous tissue, which may or may not contain small aggregations of similar cells, or even be separated by a definite layer of them from the healthy liver substance. The presence of cellular elements is a proof that the disease was still advancing at the time of death.

It is this fibrous material which gives the cirrhotic liver its peculiar characters. The early embryonic tissue lies in the angles between the hepatic lobules, around the terminal branches of the portal vein. As it undergoes development, its separate portions coalesce. Thus they gradually form fibrous rings, surrounding the hepatic lobules, or groups of lobules. But (like all young connective tissue) the new material has a strong tendency to contract. It therefore compresses the secreting cells contained in the lobules. Some of these undergo absorption, allowing the adjacent fibrous rings to coalesce. They may thus form extensive tracts of a whitish grey colour, containing only here and there a few isolated hepatic cells. Other lobules, again, become squeezed up into the rounded yellow granules or nodules above described. The reality of the compression is evident from the fact that when a section is made through a cirrhosed liver, the yellow masses at once rise and project above the cut surface; the secreting cells in the yellow masses do not lie in definite columns, but are disarranged, so that, indeed, it is impossible to determine how many original lobules each mass severally contains.

The distribution of the blood-vessels in a cirrhosed liver is very remarkable. If the hepatic artery be injected, one finds that the new fibroid tissue which looks quite bloodless is abundantly supplied with vessels from this source. But the branches of the portal vein often appear to be almost completely obliterated. Rindfleisch says that in one case he found it impossible to force injection beyond the three or four main divisions of its trunk. He therefore supposes that in this disease the bile is mainly elaborated from arterial blood.

As a rule, the hepatic ducts are but little obstructed in cirrhosis. But isolated nodules are often found to be of a dark yellow or green colour, which is evidently due to local jaundice, caused by obstruction of the corresponding ducts.

In advanced cirrhosis, the liver is generally smaller than natural, and sometimes it is very greatly reduced in weight. I find in the post-mortem records of Guy's Hospital several instances in which the organ has weighed as little as thirty-two or thirty-four ounces, and cases have been recorded in which it has been still smaller. But not uncommonly a cirrhosed liver is found after death to be of the natural size, or even above it. Whenever considerable enlargement is present, I believe that the organ is almost always also loaded with fat. We have had one remarkable instance in which a liver, which was of gristly hardness, weighed nine pounds; it contained so much fat that it would actually float in water.

It is, however, a question whether a minor degree of enlargement of the liver does not constantly occur at an earlier stage of cirrhosis. That this should be the case is evidently quite consistent with what we know to be the pathology of the disease. Unless the wasting of the hepatic cells goes on from the very first, *pari passu* with the growth of new fibroid tissue, the organ cannot but increase in size. Bright long ago stated that he had traced the enlargement of the organ when cirrhosis was beginning, and its gradual diminution towards the more confirmed stages of the affection. Indeed, it is clear that the progressive reduction of the liver to the weight of two pounds must be attended with a corresponding decrease in the area of percussion-dulness over the organ. But, as a matter of fact, I believe that the physician very rarely has an opportunity of observing this. Cirrhosis can seldom be diagnosed with certainty until it has reached an advanced stage; and the abdomen is then often so distended as to prevent an accurate determination of the size of the liver.

Thus I think it has not been proved that an appreciable enlargement of the liver is always present at the commencement of the disease. On the other hand, it is certain that fatal effects often manifest themselves at a time when the organ is still not much below the natural size, and even when it is greatly larger than natural. When one finds a patient with a large granular liver it is a grave error to suppose that his prospects are necessarily any better than if the liver were small.

Ætiology.—The main *cause* of cirrhosis of the liver is almost universally believed to be the abuse of alcoholic liquors. The terms "hobnailed liver" and "gin-drinker's liver" have long been accepted in this country as synonymous. And Dr Dickinson found that this disease was present in 22 out of 149 persons whose trade it had been to make or sell or carry stimulants, and who died in St George's Hospital; while it occurred in only 8 out of 149 other persons who had been unconnected with the liquor traffic, and who therefore may be presumed to have included a smaller number of the victims of intemperance. It has been objected that the disease is sometimes seen in children; but recent observations appear to show that this may really be an argument in favour, rather than against, its alcoholic origin. Dr Wilks some time since had a little girl, eight years old, under his care at Guy's Hospital suffering from what proved to be a very small hobnailed liver; she had been addicted to drink, having taken as much as half a pint of gin daily. And Niemeyer quotes from Wunderlich two very similar cases which occurred in sisters, aged eleven and twelve years respectively, who had each drunk spirits to excess. In fact, I believe that the disease is very rarely indeed met with among those who are known to have been temperate in their habits. It has been supposed that other ingesta besides alcohol may set up this form of chronic inflammation in the liver; and the excessive use of spices and other condiments has sometimes been charged with causing it. Niemeyer records a case in which he supposed that it was set up by the presence of gall-stones in the hepatic ducts; and I have already stated that persistent jaundice from obstruction of the biliary passages appears more or less constantly to lead to an overgrowth of the connective tissue in the portal canals—in fact, to a slight degree of cirrhosis. Obstructive heart disease, again, was once believed to be a cause of cirrhosis; but it is exceedingly doubtful whether this ever by itself gives rise to the affection, although, by retarding the circulation through the liver, it may probably enable comparatively moderate quantities of alcohol to produce it. Persistent intermittent fever is another condition that has been supposed to be followed by cirrhosis of the liver, but Frerichs seems to be right in thinking that this cause does not operate by itself. The idea that syphilis may be one of the causes of the affection may now be dismissed; for the hepatic disease which results from syphilis is really quite different.

In stating that excessive indulgence in alcoholic liquors is the principal cause of cirrhosis, however, one cannot deny that the affection is occasionally met with in children and others who are very unlikely to have been intemperate. And the late Dr Anstie, in investigating certain other effects of chronic alcoholism, found that in the immense majority of his cases there was no marked symptom of disease of the liver, nor did such symptoms show themselves, although he watched the patients for a considerable time. He therefore inferred that the abuse of stimulants could not be the cause of cirrhosis. But in drawing this conclusion Anstie cannot have allowed sufficient weight to the fact, of which he was doubtless aware, that this disease often reaches an advanced stage without manifesting itself by definite symptoms. At Guy's Hospital, the liver is accidentally found cirrhosed (in persons who have died of injury or of some other disease) once for every two cases in which cirrhosis has been the cause of death. In many of these cases, the organ is indurated in an extreme degree, yet the patient certainly suffered from none of the more marked symptoms of such an affection. I have therefore little doubt that in many of Anstie's cases the liver was really more or less cirrhosed.

The frequency with which cirrhosis of the liver is thus latent is, in fact, one of the most remarkable features of the disease; but I must add that there has been hardly a single instance in which the organ has been found, under such circumstances, to weigh less than fifty ounces. This might be taken as showing that the affection was not really so advanced as it appeared to be, and that if life had been prolonged it would have progressed and the characteristic symptoms would have developed themselves; but the cases appear to me to have been too numerous to admit of such an explanation. Moreover, on casting up the ages of persons in whose bodies cirrhosis of the liver was discovered without there having been marked symptoms during life, I find that the average age was higher by about five years than that of those persons who died of the effects of the disease. If this fact can be relied upon, and if it should be confirmed by a wider experience, I think it would prove that cirrhosis is not always (as we have been inclined to suppose) a progressive disease, but rather that after having reached a certain degree of development it often remains stationary, and remains so during the rest of the patient's life.

As regards the age of patients who die of the effects of cirrhosis of the liver, I find that between forty and fifty years of age there is a larger number than in any other decennial period. The proportion of males to females appears from the records at Guy's Hospital to be 102 to 26, or very nearly as 4 to 1.

Symptoms.—Even when cirrhosis of the liver is about to give rise to effects that will rapidly endanger life, its early symptoms are exceedingly indefinite. They are chiefly those which have already been described as indicative of "congestion" of the liver (p. 253); often, indeed, with the addition of the other symptoms of chronic alcoholism; restlessness at night, tremor of the tongue and hands, irritability of the bowels, &c. A knowledge of the patient's habits often suggests a suspicion that the liver may be cirrhotic at a time when there is no positive proof of it.

The digestive disorders which thus usher in the more serious effects of cirrhosis may be in part due to the impairment of the functions of the liver, consequent on the extensive destruction of its parenchyma. But another and perhaps a more important cause of these symptoms is the interruption of the flow of blood through the portal system of vessels, resulting from the presence of the new fibrous tissue which is developed in the substance of the organ. Pathologists have, indeed, long found a difficulty in explaining how the blood returns from the chylopoietic viscera when the liver is affected with any considerable degree of cirrhosis. Some of it no doubt escapes through the anastomoses which exist at the upper and lower limits of the distribution of the rootlets of the portal vein. Thus, the œsophagus is often found to be surrounded by a plexus of dilated vessels, which had carried upwards a part of the blood from the stomach; and hæmorrhoids are very frequently present, which may be taken as an indication that some of the blood from the rectum had passed away into adjacent branches of the iliac veins. But these communications seem quite insufficient to make up for the great obstruction that must exist in many cases of cirrhosis. Rindfleisch states that in one extreme instance, which he investigated, the portal blood passed directly into the inferior cava through a number of dilated anastomoses between the mesenteric and spermatic veins. Frerichs lays stress upon the existence of vessels in the newly-formed adhesions between the liver and the diaphragm and abdominal wall. He also adopts the statements of Sappey with reference to certain accessory branches of the portal vein, the chief of which run along the round ligament of the liver to reach the under surface of the diaphragm. In two cases of cirrhosis Sappey found this vessel distended to the size of the little finger. Some years ago I also observed a large vein in this position when I attempted to inject the portal vein in the body of a patient whose liver was hobnailed. Whatever may be the precise course taken by the portal blood, there seems to be no doubt that much of it gets into the veins which ramify over the abdominal walls, and pass upwards into the internal mammary veins. For the superficial vessels of the abdomen become greatly over-distended in cases of cirrhosis, and this can be explained in no other way than that which I have suggested—unless it could be shown that the trunk of the inferior cava itself were greatly compressed, and of this there appears to be no evidence whatever.

The congestion of the portal system of vessels which thus results directly from cirrhosis of the liver accounts for the fact that after death from this disease the stomach is generally found to be reddened and lined with mucus, and that the spleen is often enlarged. There has, indeed, been some difference of opinion about the state of the spleen. According to some writers it is almost invariably increased in size; whereas others have said that this is seldom the case. Frerichs found that the spleen was enlarged in exactly half his cases; and this statement has been adopted by most subsequent writers. Another frequent effect of the portal congestion in cirrhosis of the liver is hæmatemesis, (see pp. 131, 134).

It must be added that congestion and dilatation of blood-vessels is not limited in this disease to the radicles of the portal vein, but occurs likewise in distant parts of the body, where it is very much less easy of explanation. Thus a very frequent, and really a valuable, symptom of cirrhosis of the liver is the presence on the patient's cheeks of a number of minute red lines and points, consisting of minute cutaneous vessels that have become varicose. I am not now referring to any form of the affection known as acne-rosacea, although this too is often the result of intemperance, but to what are termed "stigmata." Besides the face, they may often be found on the chest and abdomen. Hæmorrhages from the different mucous membranes and purpuric spots upon the skin are also frequent effects of cirrhosis of the liver.

Hitherto, I have made no reference to the really important effects of cirrhosis, which, when they once develop themselves, commonly quickly destroy the patient. They may be grouped under three different heads—1, jaundice; 2, cerebral symptoms; 3 ascites. Of these, ascites is by far the most frequent and important, but on that account it will be convenient to take it last.

The production of *jaundice* by cirrhosis of the liver and its characters have already been mentioned at p. 270, where I stated that among 130 cases in which the liver was found after death to be hobnailed, there were thirty-four in which more or less jaundice existed, and nineteen in which it was intense. This statement, however, hardly does justice to the frequency of icterus, in comparison with the other symptoms of cirrhosis, for in more than forty of the 130 cases the cirrhosed state of the liver was accidentally discovered in the post-mortem room. This would leave less than ninety cases in which the cirrhosis produced marked effects during life; and among these the proportion of cases in which some jaundice was present would be more than one in every three. The liver is generally enlarged in these cases. In nearly half the instances which occurred in Guy's Hospital, with intense jaundice, the organ weighed more than seventy ounces; once as much as 131 ounces. It almost always contained much fat.

Cerebral symptoms, especially drowsiness and coma, have been mentioned as frequently ushering in the fatal termination in cases of cirrhosis which produce jaundice. They are also common in cases which give rise, not to jaundice, but to ascites. Moreover, when, by means of diuretics and purgatives, one is able to clear the peritoneal cavity of its fluid, the successful action of such remedies seems often to avail the patient very little, for he presently becomes stupid and unconscious and dies, although his abdomen may be perfectly flat and empty. The question has even been raised whether the removal of the fluid by medicines may not have been sometimes concerned in the production of the fatal issue. There seems to be no reason why similar cerebral symptoms should not sometimes appear in cases of cirrhosis that had not before been attended with either jaundice or ascites; but I do not know that any instance of this kind has as yet been observed. Dr Carrington has pointed out that pyrexia is not infrequently present.

In addition to the drowsiness and coma which are the chief symptoms that I have observed in cases of this kind, Frerichs mentions noisy delirium and (in one instance) spasmodic contractions of the left side of the face. I remember one patient who, although he could be partially roused, seemed to be quite unaware of being in bed and in the hospital, and, when asked where he was, always named some street in the city, where he had previously resided. This man lay for two or three weeks in a semi-comatose condition. The cause of these cerebral symptoms is, I think, as yet unknown. It has been supposed by some writers that it results from a disintegration of the secreting cells of the liver, like that which occurs in acute yellow atrophy. But I have several times carefully examined the tissue of the organ in such cases, and I have always found numerous liver-cells in an apparently unaltered state. The microscopical characters were, in fact, undistinguishable from those of any other cases of cirrhosis. Frerichs, however, states that in his cases a large quantity of leucin separated from the organ, and that the bile-ducts contained only a small quantity of pale bile. He therefore describes the condition in question as one of "acholia." Another theory, which has been propounded by Dr Austin Flint (of New York), is that the cerebral symptoms in these cases, as well as in those of acute yellow atrophy, are due to the accumulation of cholesterine in the blood. He supposes that in health one of the functions of the liver is to eliminate from the blood cholesterine, which he believes to be a waste product derived from the brain and nerves; and in a case of cirrhosis which terminated by coma he found that there was a large increase in the amount of cholesterine contained in the blood. He has therefore invented the name "cholestearæmia" for the state in which such symptoms are developed.*

Ascites.—Most frequently, however, the principal effect of cirrhosis is not jaundice; still less the supervention of cerebral symptoms. It is the presence of fluid in the peritoneal cavity; a form of dropsy which is technically termed *ascites*. This is sometimes first discovered by the physician, when there had before been no suspicion of it; sometimes the patient finds it out for himself, by the fulness and sense of weight in the belly to which it gives rise. An examination of the abdomen, however, is always required to determine the presence of fluid with certainty. For the patient may experience exactly the same sensations from the accumulation of flatus in the bowels, and of fat in the subserous tissue. A large proportion of those who seek advice for abdominal dropsy are really free from this complaint.

Palpation and percussion are both useful in revealing the presence of fluid within the peritoneal cavity.

1. *Palpation* may be employed in two distinct ways. If any solid organ or tumour lies at a little distance from the anterior wall of the abdomen, separated from it by fluid, one can often, by a sudden movement of

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* [Charcot believes that when these cerebral symptoms are present, with jaundice and without ascites, the liver will be always found enlarged; and that the cirrhotic change has then begun, not around the lobules in the portal canals, but within the lobules. He also believes that this "hypertrophic cirrhosis" is not, like the ordinary hobnailed liver, due to drink. See paper by Dr Price (with table of 142 cases), 'Guy's Hosp. Rep.,' xlii, p. 295.—Ed.]

the fingers, depress the abdominal wall, and push aside the fluid, so as to feel the solid mass beneath in a way that would be impossible if no fluid were present. Thus one may not only detect an enlarged liver, but also at the same time determine the presence of ascites. This procedure is sometimes spoken of as "dipping for the liver;" it requires a little dexterity, and should be carefully practised by the student. But a hobnailed liver which causes ascites is generally reduced in size, instead of being larger than natural (see note on preceding page). Consequently, one cannot very often arrive at a positive result in this way in cases of cirrhosis.

The other method of discovering by palpation whether there is fluid in the peritoneal cavity, is by observing whether "fluctuation" can be felt.

This term, as every student knows, is commonly employed by the surgeon to designate the peculiar elastic sensation which results from manipulation of an abscess or other cavity containing fluid. But the way in which "surgical fluctuation" is detected is not that which reveals to the physician the presence of ascites. To understand the latter, we must bear in mind the fact that the walls of the cavity in which the fluid lies are everywhere more or less yielding, and that in part they consist of thin membranes, separating it from spaces containing air. Hence, when an impulse is given to the wall of the abdomen at one spot, the fluid can transmit it freely in the form of a wave. If, for example, the left hand be placed upon one side of the patient's abdomen, and a tap be then given to the other side with the right hand, the left hand receives a distinct shock. When the parietes are thin, and other conditions favourable, the slightest touch may cause a thrill that can be felt all over the belly. There is perhaps no other physical sign which the tyro recognises so easily as this. If, however, the parietes are massive, and very hard, or loaded with fat, the detection of fluctuation may be difficult. The two hands must then be placed near one another; and a smart blow must be given with one hand, while attention is closely directed to the reception of the impulse with the other. Sometimes the fat in the abdominal walls gives a sensation that might be supposed due to fluctuation. To avoid the possibility of error from this source, one may get an assistant to hold a thick piece of cardboard between one's two hands, with its edge pressed upon the surface of the abdomen. In some cases I believe that no fluctuation can be detected, although a large quantity of fluid is present; probably the walls of the space containing it are then too unyielding on every side for a wave to be transmitted.

It is remarkable what small quantities of fluid can often be detected in the way just described. One might have expected that unless it were present in large amount it would all have gravitated into the loins or into the pelvis (according to the position of the patient), when it would have been out of reach. On the contrary, distinct fluctuation can frequently be felt over parts of the abdomen when the intestines can be proved to lie immediately in contact with the parietes. This depends upon the same principle which I have already laid down, in explaining the red lines that are observed in the bowels in acute peritonitis (pp. 238—9). When fluid is poured out into the abdominal cavity it enables the intestines to assume their natural cylindrical form; filling the angles and corners between their convolutions and the anterior wall of the abdomen.

2. *Percussion* is also of service in detecting the presence of ascites, and still more in distinguishing this from some other conditions which resemble it in causing abdominal enlargement. Whenever the amount of fluid is at all considerable, that part of the abdomen which contains it gives a dull note on percussion. But a small quantity, lying among the intestines in the way just described, may fail to affect the natural tympanitic note, and this although it gives rise to distinct fluctuation.

For the diagnosis of ascites, however, something more is required than the mere discovery of dulness on percussion, or even of fluctuation. The former might be caused by a solid tumour, and the latter might depend upon a collection of fluid within one of the hollow viscera, or in an adventitious cyst. Cystic disease of the ovary is by far the most important of all the affections that may be confounded with ascites. But there are several other conditions that, as a matter of fact, have been mistaken for it. It is recorded that John Hunter once tapped the bladder in the belief that the patient had abdominal dropsy; and Murchison relates a case in which 480 ounces of urine were drawn off by a trocar introduced midway between the umbilicus and sternum, it having been thought that there was a hydatid tumour. Such a tumour, again, when of great size, may yield physical signs more or less like those of ascites (cf. p. 326); and so may a renal cyst; and still more commonly a pregnant uterus.

Now, in the great majority of cases one can readily distinguish an accumulation of fluid in the peritoneal cavity from all these conditions, by simply noticing which parts of the abdomen are dull, and which are resonant on percussion, particularly if one makes the patient assume different positions in turn. In ascites the fluid, being specifically heavier than the intestines, tends, in the main, to sink towards the more dependent part of the peritoneal cavity; while they may be said to float in it. Hence when the patient lies upon the back the bowels fill the umbilical region and the percussion-note there is tympanitic, whereas in the flanks it is dull. But if the patient is made to turn upon one side, the position of the intestines at once becomes altered; whichever side is uppermost is now resonant, the dulness on the other side undergoing a corresponding increase. And, when the patient stands upright, the fluid gravitates towards the lower part of the abdomen, which, up to a certain level, becomes uniformly dull. Again, when in ascites the border of the dull region is percussed firmly, the left hand finger being pressed backwards as much as possible, one can often detect a resonant note, from the presence of intestine beneath.

All these characters are wanting when enlargement of the abdomen is due to cystic disease of the ovary, or to pregnancy or to distension of the bladder. Moreover, all of these rise from the pelvis into the front of the abdomen, pushing the intestines backwards. Consequently, when the patient lies upon the back, the front of the abdomen yields a dull note on percussion.

Thus, then, most cases of ascites present *positive* characters, the recognition of which renders it impossible for a mistake to be made.* Another sign of minor value is the occasional presence of a protrusion at the umbilicus, containing fluid. But it sometimes happens that an enlargement of the abdomen is really due to the presence of fluid in the peritoneal cavity, and yet that the signs which are distinctive of ascites are wanting, the whole of the front of the abdomen being dull, in whatever position the patient may lie. This may arise in two ways. When the quantity of fluid is very large, the intestines may stretch the mesentery to its full extent, and yet be unable to reach the anterior abdominal wall. But it is probable that this very rarely occurs, at least while the parts concerned are in a normal condition. In the immense majority of cases, when the anterior part of the abdomen is dull in ascites, the reason is that the mesentery has been shortened by chronic inflammation so that it tethers the bowels closely, and prevents their floating. Or, again, the intestines may be actually fixed to the back of the abdomen by adhesions.

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* I do not make an exception for the case of an ovarian cyst containing air as well as fluid because I can hardly believe that the physical signs would then be really like those of ascites. Alteration in the position of the patient might be attended with changes in the percussion-note; but these would generally be limited to a part of the abdomen. In the instances of this kind that I have seen, a very marked splashing sound has been caused by manipulation of the abdomen: and the outline of the cyst has also been very evident.

Under these circumstances the results of percussion may be said to be *negative* so far as concerns the diagnosis between ascites on the one hand, and ovarian disease, pregnancy, &c., on the other hand. We have then to consider what are the *positive* signs of these several conditions. Now, pregnancy is distinguished by many indications, upon which I cannot enter in this place. The positive signs of an ovarian tumour are likewise in many cases conclusive. The patient may be able to say that the swelling distinctly began on one side of the abdomen. Again, a careful examination of the swelling will often lead to the detection of a solid substance in some part of it, if it be due to an ovarian tumour, or the outline of the cyst may be felt at some part of its circumference, or at least when the patient draws a deep breath a transverse line (corresponding to the upper border of the tumour) may be seen to descend. On this last sign Mr Spencer Wells lays especial stress, and the same writer also points out that whereas in ascites the greatest circumference of the abdomen is at the level of the umbilicus, in ovarian disease it is often some inches below this ; and again, that in ascites the umbilicus usually retains its natural position, being about one inch nearer to the pubes than to the ensiform cartilage, but that in ovarian disease this relation is often greatly altered.

Sometimes, however, all the positive signs of ovarian disease are wanting on the one hand, just as are those of ascites on the other hand. It may then be impossible to make a diagnosis. In such cases, when paracentesis has to be performed, the character of the fluid which is drawn off often clears up the doubt as to the nature of the disease. That which comes from an ovarian cyst is frequently viscid and of a dark greenish-brown colour, quite unlike the secretion of a serous membrane. Its viscidity is said to depend upon its containing a modification of albumen (paralbumin of Scherer) which does not coagulate when boiled with a small quantity of acetic acid. Paralbumin is said never to be present in ascitic fluid, and, on the other hand, the latter often contains fibrin, which is absent from the contents of an ovarian tumour. Thus it is said that a liquid containing both paralbumin and fibrin must necessarily have been originally secreted by an ovarian cyst, which afterwards burst into the peritoneal cavity. Ovarian fluid, however, is not always viscid, nor of a dark colour ; it may be pale yellow, and in fact undistinguishable in appearance from the fluid of ascites.

I have already mentioned that there are other affections of the liver, besides cirrhosis, of which ascites is the chief symptom. And since some of these require separate description, it will be convenient to give it now.

1. Among them I may first take *chronic inflammation of the capsule of the liver*, or, as it is often termed, *perihepatitis*. In this affection the organ is remarkably deformed. It no longer has a sharp edge, but is converted into a rounded mass. Its capsule is opaque, and often forms a separable layer, which, when stripped off, leaves a smooth surface, just like that of the healthy peritoneum. The alteration in the form of the liver is in part caused by the contraction of this thickened capsule. But very commonly its anterior edge is also folded over on to the dorsum in a way that is difficult of explanation. I lately made a post-mortem examination of a case in which the margin of the liver thus touched a part of the convex surface that should have been four and a half inches distant in a direction from before backwards. When the capsule was removed the organ returned to its natural shape. The weight of a liver affected with perihepatitis is generally about the same as that of a healthy organ. Its tissue is commonly soft, and is very often loaded with fat. It is seldom cirrhotic, but there is sometimes an excess of white fibrous tissue in the course of the larger portal

vessels. Perihepatitis appears to be a frequent cause of ascites. At Guy's Hospital there is one fatal case of it for every five of dropsy from cirrhosis of the liver; and this proportion would be greatly increased if I were to take into account those cases in which thickening of the capsule of the liver is merely a part of a general chronic peritonitis. Unlike cirrhosis, perihepatitis seems to be very rarely found in the bodies of persons who die of other diseases or are killed by accident; and from this I infer that it never remains latent, but always advances until it causes ascites. Again, in cirrhosis the kidneys are generally healthy, but in the majority of cases of perihepatitis they are diseased. It follows that, if in a case of ascites the urine be healthy, there is but little likelihood that the cause is inflammation of the capsule of the liver; and, on the other hand, that when in a case of renal dropsy the abdomen is filled with fluid to a degree disproportionate with that of the serous effusion in other parts, this is probably due to perihepatitis rather than to cirrhosis. With regard to the causes of perihepatitis I believe very little is known; according to Murchison it occasionally arises by extension of inflammation from the base of a chronic ulcer of the stomach.

2. Another affection of the liver which has to be enumerated among the causes of ascites is *simple chronic atrophy.* The 'Pathological Transactions' contain two very striking cases of this kind, in which the abdomen contained a large quantity of fluid. In one of these cases, which is recorded by Dr Cayley (1868), the liver weighed only twenty-two ounces; the left lobe had almost disappeared, being only an inch wide. In the other case, one of Murchison's (1867), the organ weighed twenty-five ounces; its margin was thin and flat, forming a kind of rim, which consisted only of connective tissue and vessels enclosed between the two layers of the capsule. This rim measured in one place an inch across. In both cases the substance of the liver was of a dark colour, and quite free from induration. Minor degrees of atrophy of the liver are by no means uncommon, particularly in old people and in those who die of wasting diseases, such as cancer of the stomach or œsophagus. But I believe this affection very seldom gives rise to any symptoms. In twenty years we seem to have had at Guy's Hospital only one fatal case in which ascites could be said to have depended entirely upon simple atrophy of the liver.

3. Again, *syphilitic affections of the liver* sometimes cause effusion into the peritoneal cavity. These present very different characters in different cases. Sometimes gummata are scattered through the hepatic tissue, which is in other respects healthy. This condition is generally unattended with any symptoms, but it may happen that one of the gummata is so placed as to obstruct the circulation through the organ, and so sets up ascites. A case of this kind occurred at Guy's Hospital, in which one of the hepatic veins, close to the inferior vena cava, was so narrowed that it would only just admit a probe. More often the gummata, instead of being embedded in the hepatic tissue, lie in the middle of broad fibrous bands, which traverse the liver from one surface to the other, forming deep notches or depressions, or cutting off large masses from their continuity with the rest. Again, beside containing gummata and fibrous bands, the liver is very often lardaceous. Now, ascites is especially apt to occur in the cases last mentioned. The organ may then reach a very great size, weighing from six to seven pounds. The capsule is very generally thickened and adherent to adjacent parts. A striking case, which appears to have been of this kind, was lately recorded by Dr Grainger Stewart. A patient had ascites, for which she was tapped twenty-one times, the enormous quantity of 12,120 ounces being removed in the course of these operations. At first the paracentesis had to be repeated every fortnight, but the intervals gradually became longer, until at length she regained a fair state of health. I find that within the last twenty years there have occurred in Guy's Hospital about six cases of fatal ascites due to

this kind of disease. In several of them the liver could be felt during life to be enlarged and adherent to the parietes, and to have an uneven and nodular surface, and these characters more than once enabled a correct diagnosis to be made.

4. *Carcinoma of the liver* is another disease that may give rise to the effusion of fluid into the peritoneal cavity, but comparatively seldom in large quantity. I am now referring to the presence of malignant growths in the substance of the organ itself, and not to the affection, already described at p. 275, in which cancer merely involves the different structures in the portal fissure. Carcinomatous growths in the liver vary widely in their clinical significance. A great many nodules may be scattered through it without producing any symptoms, and, unless they reach a considerable size, without even enabling its edge to be felt below the ribs. But when the tubera are large, they may grow to such a size that they can be felt through the abdominal walls, or even be seen to rise and fall each time the patient breathes. They are generally firm, or even of stony hardness; but sometimes they are very soft, so that one might imagine that fluctuation was to be detected in them. Indeed, their centres not rarely become hollowed into cavities containing fluid. I lately met with an instance in which a cancerous tuber formed a cyst that would have held a cocoa-nut; it was so near the surface of the organ that I think it might have yielded fluctuation; it was filled with a clear straw-coloured liquid. Sometimes cancerous nodules are rounded on the surface, sometimes one or more of them can be felt to have a central depression or umbilicus; this is of importance, for it is not observed in any other affection. Instead of many distinct nodules or tubera, cancer of the liver may form a single large rounded mass, projecting from the right or left lobe downwards into the abdomen, or upwards towards the chest. Sometimes, again, a cancerous liver is uniformly enlarged, without its shape being in any way altered. It may then reach an enormous size; two that have been observed at Guy's Hospital have each weighed eighteen pounds.* When the organ is affected with this diffused form of cancer, the appearance of its section is very peculiar. The lobular markings are everywhere plainly visible, but they are coarser than is natural. The substance of the liver is of a greyish colour, or even perfectly white; all parts of the cut surface yield a milky juice, and the microscope shows that the cells in the lobules have the character of cancer-cells, although they are arranged in radiating columns, occupying the meshes of the blood-vessels, like the secreting cells of the healthy organ. Pathologists believe that these cells are directly derived from those of the pre-existing hepatic tissue.

Cancerous tumours of the liver are often exceedingly vascular, and their vessels have very thin walls, so that hæmorrhage into the substance of the nodules is far from uncommon. According to Frerichs, such hæmorrhages may be so copious as rapidly to give rise to a perceptible increase in the size of the tumour, and even to produce general anæmia. Sometimes, again, when a vascular cancerous growth is situated just beneath the surface of the liver, the serous membrane covering it gives way, and blood escapes from its substance into the peritoneal cavity. A remarkable instance of this lately came under my observation, in which a large clot covered the surface of the organ. One can seldom determine, in cases of this kind, what quantity of blood has exuded, for much of it is mixed with the ascitic fluid; but it would seem that the fatal issue is sometimes more or less directly due to the rupture of the tumour, for patients in whom this has occurred have been observed to fall into a state of collapse some hours, or even as long as three days before death.

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* In a case recorded in vol. xxiii of the 'Pathological Transactions' a liver, which was full of cancerous tubera, weighed 19½ lbs., and another case is there alluded to in which the weight was 24lbs. Dr Arthur Jones, of Northampton, has mentioned to me a case in which the weight of 28 lbs. was reached.

In the foregoing paragraphs I have not attempted to draw a line of demarcation between primary and secondary carcinomatous growths in the liver. The truth is that the distinction is often made with great difficulty, even in the post-mortem room. All authorities are agreed that scattered nodules and tubera are almost invariably secondary. Some observers think that even the largest solitary masses, and the diffused forms of cancer of the liver, are very rarely primary. But my own impression is that such a view may be stated too absolutely. It is true that a post-mortem examination sometimes reveals the presence of a primary carcinoma in the intestine or the vertebræ or the os innominatum, which had been before unsuspected, the affection of the liver having alone been recognised clinically. But it is also true that in other cases no primary disease outside this organ can be discovered on the most careful examination. One source of fallacy may be mentioned, which is that cancer of the gall-bladder growing into the hepatic tissue has sometimes been mistaken for a primary cancer of the liver. The cavity of the gall-bladder may in such a case be so small that, lying in the centre of the tumour, it is easily overlooked.

It was shown by Frerichs that cancerous growths in the liver derive their vascular supply mainly from the hepatic artery, and that they receive very little blood from the portal vein. In proportion as they increase in size even the trunk of the hepatic artery becomes enlarged; while the area of distribution of the portal vein is diminished. The growth, however, not infrequently penetrates into the interior of one of the branches of the last-named vessel, and may then extend along its channel so as to obstruct the flow of blood through a large part of it. These facts doubtless explain the frequent occurrence of ascites in cases of hepatic cancer; but probably this is sometimes the result of chronic peritonitis which started from the serous covering of the organ. Another symptom of the disease is pain, which is often severe, and generally accompanied by marked tenderness on pressure in the right hypochondrium. Jaundice is comparatively seldom present, or shows itself only when the case is about to terminate fatally. In this respect there is a wide difference between cancer of the liver itself and cancer of the structures in the portal fissure. I have already pointed out that the latter is a frequent cause of jaundice.

5. In the various forms of disease that have hitherto been described as causing ascites this has been due to compression of the branches of the portal vein within the liver. But there is another class of cases, in which the trunk of the vein itself is obstructed.

By far the most frequent cause of this is an affection which has already been described (p. 275), as giving rise to jaundice, namely, that form of cancer which involves the gastro-hepatic omentum and the parts contained in it. I have already mentioned how often the peritoneal cavity contains fluid in this disease, and, indeed, that, except cirrhosis of the liver, it is the only affection which commonly produces at the same time jaundice and ascites. Sometimes, however, the latter occurs without the former. In 1872 I had under my care an old woman whose abdomen contained an enormous quantity of fluid, and who was tapped three times; after death the cause was found to be a cancerous growth about the head of the pancreas, by which the portal vein was so narrowed that it would only just admit a probe. A similar case had occurred in the hospital about five years before.

Another affection of the trunk of the portal vein that is sometimes concerned in the production of ascites is thrombosis, or (as it is sometimes termed) *pylephlebitis adhesiva.* This, however, is a very rare condition, and I have only seen it in association with advanced cirrhosis or capsulitis of the liver. by which the circulation through the vein had been evidently greatly interfered with before death. Frerichs states that thrombosis of the portal vein may be suspected when ascites develops itself very rapidly, particularly if the fluid should reaccumulate very quickly after tapping. But on looking through the reports of such cases which are given by this writer, I fail to make out that they establish this statement. Indeed, it appears that the flow of blood through the portal vein is often arrested by cirrhosis of the liver as completely as it could be by an actual obliteration of the vein; and it is certain that the fluid may collect again with remarkable rapidity after paracentesis, when this disease is the cause of ascites, without there being any further obstruction from thrombosis of the vessel.

Diagnosis.—It now becomes a question whether it is possible to distinguish at the bedside the various affections that may give rise to ascites by obstructing the flow of blood through the portal vein; and it appears that two of them —cancer of the liver and syphilitic disease of that organ—scarcely ever produce symptoms without at the same time causing an enlargement that can be easily detected in the right hypochondrium, or, at least, without causing a marked increase in the hepatic dulness. In perihepatitis, again, there is almost always albuminuria, due to coexisting disease of the kidneys.* On the other hand, simple atrophy of the liver and thrombosis of the portal vein occur so rarely that they need hardly be taken into consideration, and the same thing may also be said of cancer involving the lesser omentum, so far as concerns the production of ascites without jaundice. There remains only cirrhosis of the liver.

But in most cases of ascites the difficulty lies not so much in distinguishing what disease of the liver is its cause, as in determining whether it is really due to portal obstruction, or whether it arises from some affection of the peritoneum itself. I think it is generally supposed that these affections are comparatively rare; but at Guy's Hospital I find that they are very frequent, and that they include at least one third of all the cases of ascites occurring independently of heart disease or Bright's disease and unattended with jaundice.

Now, so long as the quantity of fluid in the abdomen is not very large, one can generally without much difficulty distinguish ascites caused by obstruction of the portal veins from effusion due to chronic disease of the peritoneum. In cases which come under the first head, the area of dulness in the right hypochondrium is diminished, the intestines float freely towards the anterior wall of the abdomen, there is often a history of intemperance, with the chronic disorders of the digestive organs that result from it, the face is often blotchy, and the urine is high coloured, depositing lithates stained with purpurine. In cases belonging to the second head, the front of the abdomen is very generally dull from retraction of the bowels, there may be no history of intemperance, the patient may have a clear complexion, and (in the case of malignant or strumous disease of the peritoneum) the omentum may be felt hardened and nodulated; or, again, there may be a cancerous tumour of the ovary, or evidence of cancer of the stomach, or a cancerous nodule at the umbilicus.

But whenever the abdomen is greatly distended with fluid,—so that it is universally dull in front, and yet one cannot tell whether the mesentery tethering the intestines backwards is contracted or not—the cause of the effusion is in fact beyond recognition by means of a physical examination. And it is in these very cases, when they occur in females, that (as I have already stated) it is impossible to determine with certainty the absence of cystic disease of the ovary. We have then to fall back upon the general appearance of the patient, and a knowledge of his, or her, habits; but these afford very uncertain grounds for diagnosis. When paracentesis has been performed, the nature of the fluid may throw some further light upon the question. The characters of the fluid contained in cystic tumours of the ovary have already been described (see p. 294). In cases of cancerous disease of the peritoneum, the fluid, although free from viscidity, is often of a brownish colour, or reddened, from the presence of blood. On the other hand, in cases of simple chronic peritonitis, and also in cases of ascites from disease of the liver, the fluid is generally straw coloured. In very rare cases an opaque white fluid has been removed by tapping; it has been supposed that its milky appearance resulted from the admixture of chyle, some lacteal vessel having opened into the peritoneal cavity by ulceration. A thorough physical examination of the abdomen should always be made after paracentesis; this often clears up the nature of a doubtful case, by leading to the discovery of a solid tumour, or of some disease of the liver, or of the intestines, that could not previously be detected.

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* It must be remembered that (as Murchison has pointed out) albuminuria may be caused by the pressure of fluid contained in the peritoneal cavity upon the renal veins, without there being any disease of the kidneys; but this occurs only when the quantity of fluid is large.

The *prognosis* is in almost all cases of ascites very unfavourable. Some of the diseases that give rise to it run, from the first, a course that tends inevitably to a fatal termination; and others, of which the progress is less rapid, do not cause the effusion of fluid into the peritoneal cavity until they have reached a very advanced stage. In cirrhosis of the liver, especially, I find that death occurs in the great majority of cases in from six weeks to six months after the detection of ascites. There are, indeed, exceptions to this rule. I remember the case of one patient who recovered from ascites and jaundice under medicinal treatment. He remained well for several months, after which the fluid reaccumulated and he returned to the hospital to die. He had been supposed to have some syphilitic affection of the liver, but it turned out that the disease was cirrhosis. I believe that when recovery takes place from ascites (whether after paracentesis or otherwise) the great probability is that the effusion was the result of either chronic peritonitis or perihepatitis. I have already mentioned (p. 249) that in children and young persons a form of ascites is not infrequent which is curable, even when it is the result of tuberculous peritonitis.

It may here be remarked that ascites does not always go on increasing until the pressure caused by the fluid destroys life. Sometimes the umbilicus, having been first forced outwards, gives way, and allows the contents of the abdomen to escape. Sir William Jenner has recorded a case in which (no doubt from the presence of air as well as fluid) the rupture was attended with a report loud enough to be heard at a distance from the patient's bed. The fluid may continue draining away for a time; but it rarely happens that this postpones for any long period the fatal issue.

That the *treatment* of ascites, and of the diseases that cause it, is very unsatisfactory, may be inferred from the previous paragraph. Sometimes, indeed, diuretics may be prescribed with advantage. Of these none appears to be more efficacious than copaiba; but it very often disturbs the stomach, so that the patient cannot continue to take it. The acetate or the bitartrate of potass, the spirit of nitrous ether, the compound spirit of juniper, the decoction of broom-tops, the infusion of digitalis are other remedies of approved value, and a favourite prescription at Guy's Hospital has always been a diuretic pill containing the grey oxide of mercury, powdered digitalis leaves, and powdered squill (of each a grain) which is given each night and morning. Purgatives, also, are useful, especially those which cause watery discharges from the bowels, such as the compound jalap powder. Murchison recommends an electuary composed of this powder, mixed with confection of senna; and in giving the usual advice that aperients should be taken in the morning, he lays stress upon the reason for this, namely, that otherwise the food which had been newly taken may be swept away by them, and so the nutrition of the patient be interfered with. He also insists on the caution required in the administration of drastic purgatives, lest they should set up an enteritis, which may even be fatal. My own experience fully confirms the importance of this suggestion. When diuretics and purgatives fail, trial may be made of tonics, as quinine.

Paracentesis.—In most cases, however, tapping for the withdrawal of the fluid is sooner or later necessary. It should be performed only when the distress caused by the distension of the abdomen becomes unsupportable.

The best indication that it is really necessary is perhaps afforded by the state of the breathing, which becomes greatly hurried and very shallow, from the diaphragm being pressed upwards and the lower ribs stretched. The heart also is felt beating above the nipple, and not in the usual place, but it is to be noted that this is often observed when the ascites has by no means reached a very advanced stage.

In performing paracentesis abdominis, the surgeon should use a trocar of moderate size. This is to be introduced in the median line of the abdomen, below the umbilicus, it having first been ascertained that the spot selected yields a dull note on percussion, and consequently that the intestines are not in the way. The trocar should be fitted with a long piece of elastic tubing, by which the fluid can be carried into a pail placed below the patient's bed, and the entrance of air prevented.

The operation is by no means unattended with risk, immediate and prospective. The patient has sometimes fainted, and even died, while the fluid was escaping through the trocar. It has, however, long been recognised that the cause of such an accident is the sudden removal of pressure from the viscera, and the danger is obviated by having a jack-towel folded round the abdomen before the operation is commenced, which is held by assistants, and tightened as the fluid escapes. When the fluid ceases to flow, the operator removes the cannula with one hand, while with the other he grasps the surrounding integument, so as to prevent the entrance of air into the abdominal cavity. A pad of lint is then placed over the wound, and upon this a few broad strips of plaister. But it often happens that this fails to close the opening into the peritoneal cavity. The fluid then keeps oozing out, and saturates the patient's clothing or the bed on which he lies. Such cases generally terminate fatally within no very long period.

In other instances, again, tapping is quickly followed by peritonitis which proves fatal in the course of two or three days. And even if this danger be escaped, the fluid almost always begins at once to reaccumulate, being, indeed, poured out much more quickly than before, in consequence of the absence of pressure upon the serous surface. The operation soon has to be repeated, and the patient is again exposed to the same risks as before, with an ever-increasing certainty that the relief will be but temporary and of short duration. Sooner or later he dies, exhausted by the drain of fluid, or by diarrhœa, or by hæmorrhage from the stomach or bowels.

But even though paracentesis abdominis may thus fail to prolong life, it is not therefore to be held useless. It almost always affords great relief to the patient's sufferings, and it should never be delayed when the urgency of the symptoms demands its performance.

In very rare cases, indeed, this operation is as successful as one could possibly wish. There may be no return of the ascites at all; or, as is more often the case, the fluid may be very slow in reaccumulating. Moreover, paracentesis, by relieving the kidneys and veins of pressure, may do much to assist diuretics and increase the flow of urine.

ABSCESS OF THE LIVER

Geographical distribution—Ætiology—Relation to dysenteric ulceration—Morbid anatomy—Characters of the pus—Direction of rupture—Symptoms—Physical signs—Diagnosis—Treatment by drugs—Prognosis—Paracentesis.

Allusion has already been more than once made to the occurrence of suppuration in the substance of the liver. It has been stated that a large number of abscesses are often found in cases of pyæmia, and reference has also been made to an affection in which pus is found in the portal canals throughout the organ. In neither instance, however, is any considerable quantity of matter generally developed at any one spot in the liver.

We have now to consider a very different class of cases, those in which a single abscess (or at least a very limited number of abscesses) arises in the substance of the liver, which not infrequently attains a very large size.

In England this disease rarely occurs. Indeed, some writers have stated that it is never seen, except in those who have previously lived in a hot climate, and the name of "tropical abscess of the liver" has therefore been assigned to it. This statement, however, is not altogether accurate. I find that within the last twenty years there have been in Guy's Hospital fifteen cases in which death was caused by the formation of a single large abscess (or in one instance two large abscesses) in the liver. Five of these cases occurred in persons who had come from China, or India, or the West Coast of Africa, but in ten of them there is no such history, and in the majority of them it is positively stated that the patients had never been out of England. Still it is undoubtedly true that this form of hepatic abscess seldom arises here, whereas in India and other hot climates it is very common, and indeed plays a most important part in the European death-rate In the West Indies, however, it is said to be comparatively rare.

Ætiology.—It has long been known that abscess in the liver and dysentery are often associated, and many different opinions have been held as to the connection between these two diseases. Annesley supposed that sometimes, in consequence of the existence of a hepatic abscess, the bile acquires peculiarly irritating properties, and thus sets up inflammation and ulceration of the intestine. On the other hand, Dr George Budd in 1842 propounded the theory that dysentery is really the earlier of the two occurrences, and that abscess in the liver is the result of the absorption of some morbid product from one of the intestinal ulcers; in other words, that the pathology of the so-called tropical or single abscess of the liver is essentially the same as that of the multiple abscesses with which we are familiar as arising from general pyæmia. Dr Budd's view for some time received general acceptance, but it is rejected by almost all recent writers who, from their experience of tropical diseases, speak with special authority upon this subject.

Some of the arguments which these writers use are not of any great weight. Thus it is said that if dysenteric ulcers in the colon were the cause of abscess of the liver, the same result ought to follow other forms of intestinal ulceration, such as the tubercular, or that which occurs in enteric fever. But it is well known that in all inflammatory affections the liability to the production of pyæmic infection depends essentially upon the character of the original mischief, and in dysentery this is just such as would be likely to set up blood-poisoning, which is not the case in the other two diseases that I have named.

Another argument is that many cases of hepatic abscess, in which recovery takes place, run their whole course without the patients having any symptoms of dysentery; and another, that when such symptoms do occur they often seem to follow rather than to precede those which indicate the occurrence of suppuration within the liver. But as I have said when speaking of dysentery (p. 184), it is a question whether this disease does not frequently begin insidiously, or even remain altogether latent. In England this is often the case, and it very probably is so in India likewise.

Again, it is said that abscess of the liver does not occur in all epidemics of dysentery, and is comparatively uncommon in certain countries where dysentery prevails. Aitken speaks of it as being very rare in China, as compared with India. Dr Baly did not meet with it in the epidemic of dysentery at Millbank. An answer to this objection might perhaps be found in a more careful study of the conditions under which dysentery and hepatic abscess are found to coexist. It may be that a certain length of time is required for the development of suppuration in the liver, and thus abscess may necessarily be wanting in rapidly fatal epidemics.

But the main strength of those who deny that abscess of the liver is secondary to dysentery lies in the fact that many cases have been placed on record, in which death has occurred from the former disease in a tropical climate, and in which the intestines have been examined and found to present no sign of past or present inflammation. Murchison met with a case of this kind in a European soldier in Burmah. The man had never had dysentery, although it may be noted that while he was under observation he suffered from persistent diarrhœa. He died, and an enormous abscess, holding four quarts of pus, was found in the liver, but neither the small nor the large intestines nor the stomach presented any cicatrices or trace of recent ulceration. Again, Mr Waring collected 204 cases of abscess of the liver, in exactly one fourth of which the intestine is said to have been perfectly healthy, and Dr Morehead mentions that he has notes of twenty-one similar cases.

Now, among fifteen fatal cases of large abscess of the liver which occurred at Guy's Hospital, there were three in which no sign of ulceration was found in the intestines; one of these was a tropical case. In eleven others it is expressly stated that the bowel was or had been diseased. In one case, which came under Dr Moxon's observation, there was only a minute cicatrix in the bowel so small that it might very easily have been overlooked. I am not at all sure that we can accept this as accounting for the occurrence of suppuration in the liver, particularly as such an admission would take away the ground from beneath our feet in maintaining that tuberculous and other ulcers cannot be expected to give rise to it. It is perhaps better to defer the expression of an opinion with regard to this case and the others in which no indication of intestinal mischief has been discovered, until further researches shall have shown whether or not dysentery is apt to occur in a latent form in the East. If this should prove to be the case there would be no difficulty in accounting for an occasional case of hepatic abscess with no post-mortem evidence of ulceration of the bowel, by supposing that the latent inflammation subsided before any actual breach of surface occurred, which undoubtedly is often the case. For my own part I am very unwilling to give up the theory that abscess of the liver is secondary to dysentery until some facts shall have been adduced which show that this theory is untenable. The positive observations which support it are very strong; in this country dysentery and hepatic abscess are each so rare that their frequent association would be a most extraordinary circumstance, unless they are causally connected. And it is the rule that the ætiology of a disease can best be studied, and its origin best unravelled, in the countries where it is not too common.

In India the current opinion at the present time would appear to be that dysentery and abscess of the liver are really common results of the same causes. It is supposed that inflammation extends from the mucous membrane of the alimentary canal to the largest gland which opens into it, just as it does from the urethra to the testes in cases of gonorrhœa. The principal objection to this view appears to me to be that, while it would perfectly account for the occurrence of general hepatitis, it affords no explanation at all of the formation of a circumscribed abscess.

If pre-existing ulceration of the intestine be rejected as a cause of hepatic abscess, it is very doubtful whether any other cause can be assigned in its place. Local injury—such as a blow in the right hypochondrium—has sometimes been accused of having set up suppuration in the liver, and probably with justice, but this explanation is applicable to very few cases. Elevation of the temperature is another supposed cause, and Dr Morehead thinks that it may sometimes explain the occurrence of hepatitis during the hot months of the year in plethoric individuals who have not long arrived in India. But it appears from the statistics of the European General Hospital in Bombay that the admissions of patients with hepatitis are relatively more numerous during the months which follow the cold season, and during the cold months themselves, than during the hot months. Hence if heat be concerned in the production of abscess of the liver it can be only as a predisposing cause, but exposure to cold may perhaps sometimes be an exciting cause. There is no reason to believe that intemperance has anything to do directly with the causation of the disease.

Anatomy.—The pathological processes which are concerned in the production of abscesses of the liver have been minutely studied by German observers, and are fully described by Rindfleisch (vol. ii, p. 99). I cannot quote his account in detail here, but I may mention that he distinguishes a "thrombotic" from an "embolic" variety. In the former the inflammation is said to occupy especially the walls of the interlobular branches of the portal vein. These, and the sheaths of connective tissue round them, are swollen by an infiltration of leucocytes, and the columns of hepatic cells become compressed and perish. The adjacent masses of infiltrated connective tissue then come into contact, and they form small white granules, which are very similar in size to lobules, and which, indeed, might be mistaken for them. These "pseudo-lobules" in their turn melt away, and an abscess-cavity is formed. But fresh pseudo-lobules are constantly making their appearance at its periphery and give a ragged character to its inner surface. In the "embolic" variety, on the other hand, the portion of hepatic tissue which corresponds to the distribution of the plugged vessel becomes intensely congested; the circulation in it is entirely arrested; it sloughs *en masse*. The lobules round it undergo reactive inflammation, they become enlarged, and those nearest the sloughing part are permeated by numerous pus-corpuscles which lie outside the secreting cells, between them and the capillaries. The hepatic cells appear to take no active share in the formation of pus. It must be added that the observations on which these statements rest appear to have been made in cases of the small multiple abscesses which are not now under consideration, but it is probable that they may be applied generally.

The characters of abscesses of the liver vary greatly in different cases.

When of recent formation and rapid growth they may possess no limiting membrane whatever, the pus lying in an irregular cavity formed by reddened and softened hepatic tissue. If they have been of somewhat longer standing they are lined with a layer of opaque yellowish material, the formation of which has been described above. And very old abscesses have a dense fibrous wall, which may be three or four lines in thickness, and so hard as to feel like cartilage, or which may even be more or less completely calcified. Sometimes one finds in making an autopsy, that such an indurated wall has become more or less detached from the surrounding tissues, and lies bathed in pus which has penetrated it and is limited only by the substance of the organ. In such cases, no doubt, the abscess has been of long standing, but has undergone rapid extension shortly before death.

There have been some discrepancies in the statements of writers as to the appearance of the pus in a hepatic abscess. Dr Budd states that it is usually white or yellowish and free from odour; and no doubt this is correct. I find, however, in the records of post-mortem examinations of Guy's Hospital three cases in which it is said to have been greenish, and either mucoid or curdy. In at least two instances it is said to have been of reddish or reddish-brown colour. This is of some interest because Dr Budd has asserted that the pus of an abscess in the liver is never red, so long as it is confined within the cavity of the abscess itself, but acquires such an appearance only when it is expectorated through the lung. He regarded this kind of expectoration as affording conclusive evidence of the existence of a hepatic abscess. But Dr Morehead, who at one time held the same view, says that in more than one case of asthenic pneumonia he has observed sputa having precisely the same characters. In one case of hepatic abscess that came under my care in Guy's Hospital some years ago the fluid removed by the trocar was of a brickdust colour, looking not unlike anchovy sauce; and it had a most peculiar nauseous odour which I should certainly recognise were I to meet with it again.

An abscess of the liver may sometimes attain an extraordinary size. I have notes of one which held six pints; and Dr Maclean mentions one in which the quantity of fluid was altogether nineteen pints.

As might be expected, when the pus has reached the surface of the liver, it may make its way in various directions. Thus it may point externally in the right hypochondriac and epigastric regions, the serous surface of the liver having first become adherent to the wall of the abdomen. Or it may rupture into the peritoneum. Or, again, it may discharge itself into the stomach, duodenum, or colon. When this occurs the patient sometimes vomits a considerable quantity of matter, or passes it per rectum. But in many cases this cannot be traced. Sometimes, again, the pus burrows towards the lumbar region, as in a case which came under my observation. I have already mentioned that hepatic abscesses not infrequently perforate the diaphragm, so that their contents are evacuated through the lung, the base of which organ becomes in fact converted into a ragged cavity. With reference to this, Dr Morehead gives the caution that one must not suppose that the abscess has made its way into the chest in every instance in which a patient suffering from this disease expectorates pus, as he has seen three cases in which this symptom had been present, but in which no perforation could be discovered after death.

Symptoms.—It must next be asked—What are the symptoms which indicate the formation of a hepatic abscess? And in attempting to answer this question, the fact must be prominently brought forward that in many cases abscess of the liver remains entirely latent and is found after death in the bodies of those who had never been known to suffer from any symptoms of the disease. Cases of this kind are mentioned by all writers on tropical diseases, for it is chiefly in persons who have been in hot countries that latent abscess of the liver has been met with. Some time ago I examined the body of a gentleman who died of protracted diarrhœa a few years after his return from China. There was an abscess the size of a walnut in the posterior part of the liver, although careful inquiry during the patient's life had failed to elicit any evidence that the liver was otherwise than perfectly healthy.

Again, an abscess of the liver, which up to a certain point has gone on increasing in size without affecting the patient's health, may suddenly give rise to the most serious symptoms. Dr Maclean mentions the case (occurring in the Mauritius) of a man, apparently in good health, who had walked seven or eight miles in search of a fresh engagement, when he complained of pain at the pit of the stomach and in a few hours died; an abscess of the liver, lined with a firm cartilaginous membrane, had burst into the pericardium. Another case is that of a man who had been invalided from India on account of "chronic hepatitis," but who when he arrived at Fort Pitt had apparently recovered so completely that he was sent to the depôt for duty. Some weeks afterwards, while he was straining at stool, "something gave way;" and this proved to be a hepatic abscess, which had likewise ruptured into the pericardium. Dr Maclean supposes that the thick capsule which most of these latent abscesses possess prevents their causing constitutional irritation, by forming a barrier, so to speak, against the disturbing influence of the pus. But the growth of a dense capsule is of course the work of time; and the abscess in these cases is latent from the very first, before any capsule exists.

In many cases, however, abscess of the liver gives rise to symptoms which are strongly indicative of its presence; and the results of physical examination of the hepatic region may be almost conclusive. The symptoms are said to be a sense of fulness and weight, or even pain, in the right hypochondrium, pain in the shoulder, inability to lie on the right side, fever, disturbance of the digestive organs, and cough. The pain which attends abscess of the liver is exceedingly variable in degree. It is often much more intense in the shoulder than in the hypochondriac regions. Dr Budd mentions a case in which the pain in the shoulder was for a long time most severe; but when the abscess was opened the pain became relieved. The occurrence of pain in this spot has been said to be a proof that the disease is situated in the *right* lobe of the organ. The pain over the liver itself is often increased by pressure, and also by the patient drawing a deep breath, or turning over on to the right side.

Cough, again, is often present in cases of hepatic abscess. It is generally short and dry; and is no doubt due to reflected irritation. It might be expected that fever would be a marked symptom; and Dr Maclean states that in every case of suppuration of the liver that had recently been under his observation at Netley the thermometer showed a rise of from one to three degrees. But there were, no doubt, cases in which the existence of abscess was suspected on other grounds; there appears to be no reason for supposing that thermometric observations would often reveal its presence if it were undiscoverable by other means. Dr Morehead points out that rigors are not of much value as indicative of suppuration in the liver; they may be present when there is no abscess; and, on the other hand, they are often absent where an abscess is really being formed. Jaundice appears never to arise in the disease under consideration, unless by accident, from the occurrence of pressure upon one of the main ducts.

The *physical signs* of hepatic abscess depend mainly upon its position and its size. Twining, of Calcutta, believed that even a deep-seated abscess often indicates its presence by inducing a peculiar rigidity of the upper part of the rectus abdominis muscle on the right side. And there is no doubt that this condition of the muscle is an important sign of disease in some one of the viscera immediately subjacent; but later observers have by no means confirmed the opinion that it points especially to the disease now under consideration. The existence of enlargement of the liver is of much more diagnostic value when present; and particularly if the outlines of the organ be altered, if there be bulging or tumefaction in one particular direction, and if any spot can be detected which is soft and fluctuating. Where enlargement of the liver can be clearly made out there is generally much tenderness on pressure in the hepatic region; and round any fluctuating point there is almost always a marked inflammatory induration of the abdominal parietes.

These decided indications of hepatic abscess are of course absent in all cases in which the back part of the liver is the seat of the disease. Even then, however, there may be an increase in the area of hepatic dulness, which, if well-marked symptoms be present, may make the diagnosis sufficiently clear.

Diagnosis.—That the detection of hepatic abscess is often very difficult may be inferred from the statements that have already been made as to the vague character of the symptoms which alone are present in many cases.

According to Dr Maclean, there is another affection of the liver which is common in India, and which is very apt to be mistaken for suppurative hepatitis, viz. acute inflammation of the capsule of the liver, or perihepatitis. The symptoms of this disease, however, are said to resemble those of pleurisy rather than those of suppuration in the parenchyma of the liver. The pain is sharper and more acute than in the last-named disease, and it is more decidedly aggravated by pressure, or by a full inspiration, or by movement of the patient's body. Acute perihepatitis is probably not very uncommon in this country, for the liver is often found after death to be fixed to the diaphragm by adhesions which appear to correspond with those which would be left by an acute, rather than with those that would have resulted from a chronic, inflammation; but I do not know that this affection is at present capable of clinical definition.

Even where there are distinct signs of the existence of an abscess in the right hypochondrium, one must not take for granted that this is seated in the interior of the liver itself. It may be embedded in the substance of the abdominal walls. Again, I published in the 'Guy's Hospital Reports' for 1874, a series of cases, six in number, in which a collection of pus existed between the upper surface of the liver and the abdominal parietes. This affection is most frequently caused by direct injury to the part, or by extension of inflammation from disease of one of the other abdominal viscera. In these respects it differs from abscess of the liver itself, which, however, in all other points resembles it very closely, and probably is not capable of being distinguished from it by physical examination. A correct diagnosis between these two diseases is not a matter of merely scientific interest; for a subdiaphragmatic abscess can probably be more readily cured by evacuation of its contents than one embedded in the liver, since its walls can more easily come into apposition.

Again, a suppurating hydatid of the liver may easily be mistaken for simple abscess, if the case be first seen when inflammation has already been set up within the capsule of the cyst, and if the existence of a painless tumour previously should have escaped the observation of the patient. Indeed, in such cases it is really impossible to determine the nature of the disease until the contents of the abscess are discharged, so that they can be submitted to careful examination. Lastly, it is said that one ought to bear in mind the possibility that a suppurating gall-bladder might be mistaken for an abscess situated within the substance of the liver; but this applies only to abscesses situated in a particular region.

Prognosis and treatment.—The treatment of a case of acute hepatitis, in which suppuration is feared, must vary according as there is or is not reason to believe that pus has already been formed within the liver. Several writers of large experience in India concur in stating that in the early stage of the disease ipecacuanha is a very potent and valuable remedy. Dr Maclean says that this drug is nearly as efficacious in suppurative inflammation of the liver as in tropical dysentery, and that it should be given in the same manner, viz. in doses of from fifteen to twenty grains repeated at intervals of five, six, or eight hours. Antimony is also used by some medical men in India, either in addition to the ipecacuanha or separately; and stress is also laid upon the importance of relieving pain and giving sleep by the hypodermic injection of morphia. Fomentations or poultices are kept applied to the hepatic region.

It appears to be established that under such treatment patients often quickly recover after having had symptoms exactly like those which are recognised as the early symptoms of hepatic abscess; but I do not know whether it is certain that suppuration would really have occurred in these cases if left to themselves.

When there are grounds for believing that an abscess has actually been formed in the liver the main question as regards the treatment is of course whether or not the pus should be evacuated by the surgeon. The answer to be made to this question must depend upon the results of experience with regard to the natural course of the disease when left without interference. Now, it is certain that hepatic abscesses sometimes subside spontaneously. In making post-mortem examinations dried-up abscesses have sometimes been found with cheesy contents. I believe, indeed, that this occurs much more rarely than was formerly supposed; for, on the one hand, dead hydatids have probably often been mistaken for cured abscesses; and, on the other hand, it is certain that the same view was generally taken of syphilitic growths in the liver before the real nature of them was understood. And I think that the spontaneous subsidence of a hepatic abscess without discharge of its contents can rarely be anticipated in any case in which well-marked symptoms of suppuration have once shown themselves; and particularly if its site is indicated by definite physical signs.

Cases in which a hepatic abscess has discharged its contents spontaneously not infrequently terminate favourably, the cavity after a time closing and pus ceasing to be formed. It would appear that the prospect of such a termination has hitherto been greatest in those cases in which the abscess has made its way through the lung. Dr Stovell is said to have reported nine recoveries out of sixteen cases of this kind; but according to Morehead, there is reason to believe that the abscesses were small. Again, recovery is by no means infrequent when an abscess discharges into the stomach or intestine. Morehead had five cases which took this course; and three of them terminated favourably. Rupture into the peritoneum or pericardium is almost invariably fatal within a short space of time; but abscesses of the liver fortunately very rarely take either of these directions.

Again, when an abscess points towards the exterior of the body, and presently breaks and discharges its contents through the skin, the patient sometimes ultimately does well. Dr Maclean says that this is much more likely to occur when the point at which the abscess reaches the surface is near the ensiform cartilage than when it is in the right hypochondrium, or in an intercostal space. It appears that Morehead was the first to draw attention to this distinction, which apparently depends upon the circumstance that in the former case the collection of pus is usually a small one, being seated in the left lobe of the liver.

No better indication could be given of the extreme divergence of opinion which has existed with regard to the propriety of surgical interference in cases of hepatic abscess than the fact that some authorities, including Dr Budd, have expressed the opinion that even when an abscess is actually pointing it should be allowed to break of its own accord.

Within the last few years, however, the whole aspect of this question has been changed by the introduction of the aspirator of Dieulafoy, and and of the various appliances of the antiseptic method. No better illustration of the advantage of treating cases of hepatic abscess with the aspirator could be given than a case which is reported in the 'Medical Times and Gazette' for April, 1874, from the Madras Hospital.

An Englishman was admitted with a tender swelling extending from the hepatic region downwards to the level of the umbilicus. He had daily accessions of fever, and other symptoms indicative of deep-seated suppuration. As there was fluctuation in the centre of the swelling this was tapped with the aspirator, and four ounces of pus were withdrawn. The relief, however, was but partial; and when the operation was twice repeated only a small quantity of matter came away each time. The patient's condition kept getting worse. The existence of a second abscess was therefore suspected; and as there seemed to be a slight bulging of the right lower ribs, it was determined to make an exploratory puncture in this position. The needle of the aspirator was introduced, and the syringe was soon seen to fill with pus. Forty ounces were withdrawn; and in a few weeks the patient left the hospital well.

Such a case as this contrasts strikingly with those which are given by the older writers on the diseases of India as showing the danger of opening hepatic abscesses. If the aspirator should not be at hand, an ordinary fine trocar might probably be used quite as safely, provided that the carbolic spray were used, and the other details of the antiseptic method were attended to. It is now fully established that the introduction of an instrument of this kind into the substance of the liver is not of itself attended with any risk. When an opportunity arises of examining the parts soon after the performance of such an operation one can scarcely ever discover the track of the trocar. But it is a strong objection to puncture an abscess without an aspirator, that the pus is very likely to refuse to flow through the cannula.

This occurred in a case which was under my care in 1875; and it was not until suction was made that it began to escape. Eleven ounces of viscid reddish pus were then drawn off; the man had not the slightest elevation of temperature afterwards, and was kept in bed only as a matter of precaution. The tumour altogether disappeared. I may note that he had been in foreign service as a mariner off the Indian and West African coasts. But he had bought his discharge four years before he came into the hospital, and since then had been working as a carman. It appeared that he had suffered severely from diarrhœa, but had not had dysentery.

Even when the trocar does not reach any pus, marked relief may be afforded by its introduction into an inflamed liver. In a case of Dr Ralfe's at the Dreadnought Hospital ('Lancet,' ii, 1876), in which hepatitis came on in the course of an attack of dysentery, only a small quantity of blood flowed into the aspirator syringe, but the patient declared that he felt instantly relieved, and did in fact experience no more pain in the right hypochondrium, where it was before severe; and his temperature, which had ranged from 99·4° to 102·2°, fell in two days to normal.

PAINLESS ENLARGEMENTS OF THE LIVER

Hypertrophy—Leuchæmia—The fatty liver: of phthisis, of obesity—Treatment of obesity—The lardaceous liver—Hydatid tumour: natural history: diagnosis: events: prophylaxis and treatment.

I have already had occasion to mention several forms of hepatic disease in which the liver becomes much larger than natural, but these have been characterised by many important symptoms beside the mere enlargement. In the present chapter I propose to deal with certain affections in which an increase in size of the liver is often the only indication that the organ is otherwise than healthy. In particular these affections are unattended with pain, and Murchison has therefore conveniently grouped them together as "painless enlargements" of the liver.

1. *Simple hypertrophy.*—Some time ago I made a post-mortem examination of a case in which death occurred three weeks after an accident. The liver was found to project four inches below the ribs, and it weighed 130 ounces. I could discover no morbid change in the hepatic tissue, and I was obliged to set the case down as one of simple hypertrophy of the organ. This affection is recognised by writers on diseases of the liver, but at present nothing definite is known about it.

2. *Leuchæmic enlargement.*—In leuchæmia the liver often becomes considerably enlarged, but never, I believe, so as to give rise to any marked physical signs in comparison with those caused by the splenic tumour. This affection will be described in the next chapter.

3. *The fatty liver.*—Another condition in which the liver becomes enlarged, without pain or other marked symptoms, is that in which its cells are loaded with fat. I have notes of one case of this kind in which the organ weighed 112 ounces, and of another in which it weighed 155 ounces, or about three times its normal weight. The 'Pathological Transactions' contain a case in which it weighed twelve pounds. I have already (at p. 290) alluded to the fact that a cirrhosed liver often contains much fat, particularly when it is increased in size, and that the nodules on its surface can then often be felt through the abdominal walls. But in the affection with which I have now to deal the organ is usually perfectly smooth. Its edge is somewhat rounded. In the dead body it is found to be anæmic, having a more or less bright yellow colour, but, as Rindfleisch remarks, one must not suppose that it has the same appearance during life, for it can be injected without the employment of any great force, so that the *vis a tergo* of the blood probably suffices to overcome the resistance excited by the distended cells so long as the heart is beating. The organ is soft and tears very readily beneath the pressure of the finger. Its specific gravity is diminished, sometimes even to such an extent that it floats in water. When it is cut into it greases the surface of the knife, especially if this be warmed.

The microscope shows that the accumulation of fat takes place within the hepatic cells, and especially in those which lie towards the periphery of the lobules. These often contain drops of oil, so large as to obscure their walls, and an inexperienced observer may suppose that the cells themselves have undergone destruction.

As might be expected, one can often easily detect at the bedside a fatty liver. It may be found as low as the umbilicus, or even lower, and the smooth, even surface and the soft doughy feel of the edge can be recognised as distinguishing this from other enlargements of the organ. The deficiency of resistance may, indeed, be so great that the liver slips away beneath the hand; one may then have great difficulty in actually feeling it, even though the parietes may be perfectly soft and yielding, and percussion may indicate that the organ is much increased in size. This very difficulty, however, points to fatty disease of the liver as the cause of the enlargement.

The conditions under which the liver is apt to become loaded with fat are numerous, but they may be divided into two main classes, strikingly opposed to one another. In one of these an excess of fat is present in the body generally; in the other there is emaciation, often in an extreme degree.

The first kind of fatty liver occurs chiefly in persons who lead sedentary lives, and who eat large quantities of rich food, particularly if they also indulge freely in stimulants. This is evidently analogous to the affection that is artificially produced in geese by the purveyors of the *pâté de foie gras.* The birds are kept in a dark place, with but little space to move in, and are crammed with a paste made of farinaceous food. The consumption of fat within the body is thus reduced to a minimum, while its formation is perhaps increased. Its accumulation in the blood probably precedes its deposition in the hepatic cells.

The other kind of fatty liver cannot be so easily explained. Cases of pulmonary phthisis are those in which it most frequently occurs. I believe that this was first noticed by Louis, who found it in one out of every three bodies of those who had died of consumption. At first sight one might be inclined to attribute this to deficient oxidation of fat from interference with the respiratory function. But if this supposition were correct, the liver ought to become fatty in cases of asthma and of emphysema likewise, whereas the other diseases in which it really becomes so are such as resemble phthisis in being attended with wasting, namely, cancerous affections, ulcer of the stomach, chronic dysentery, &c. One is therefore driven to assume that in the course of progressive emaciation the blood becomes in some way overloaded with fat, which is forthwith stored up in the liver. Dr Wilks is inclined to connect the affection under consideration with the circumstance that the patient has generally been bedridden for a long time before death. As I have alluded to the *pâté de foie gras* in relation to the other form of fatty liver, I may mention that, according to Larrey, it is possible by keeping geese shut up in close, hot cages, without food of any sort, to induce a fatty enlargement of the liver while the birds themselves become greatly wasted.

As I have already stated, cases of fatty liver present neither pain nor jaundice. It is, indeed, said that when this affection is present in an extreme degree the fæces are pale, that in all probability the amount of bile secreted is much diminished, and that a sensation of fulness in the right hypochondrium may be experienced by the patient. Many years ago Addison expressed the opinion that a symptom, perhaps pathognomonic of the affection, was a peculiar state of the skin, which he described as looking semitransparent and pale, somewhat like fine polished ivory, and as feeling exquisitely smooth and soft, so as to resemble the softest satin ('Addison's Works, Syd. Soc., p. 102). I believe that this is observed in the form of fatty liver which accompanies emaciation. On the other hand, when the patient is the subject of obesity, the skin acquires a shining, greasy appearance, apparently due to an excessive secretion of fat by the sebaceous glands.

It is said that when such persons sweat, the fluid is unable to wet the skin and runs off in large drops. Some writers have said that diarrhœa frequently depends upon fatty disease of the liver. They have, in fact, shown that it is present in many cases of this kind, but perhaps they are both effects of one cause, instead of standing to one another in the relation of cause to effect.

In a case of phthisis or other wasting disease the detection of a fatty liver does not affect the treatment, except that probably cod-liver oil and the like should no longer be given. It even influences the prognosis but little, since such cases are generally fatal, and the most that can be inferred from it is the fact, already sufficiently apparent, that the patient's nutrition is greatly damaged. On the other hand, when the same affection is part of a general obesity, it is this, and not merely the state of the liver, which calls for interference on the part of the physician.

Still, as I shall have no other opportunity of discussing the treatment of obesity, I must devote a few lines to it, and it must be distinctly understood that this condition is itself one of grave import. No good insurance office will accept at ordinary rates the life of a man whose weight bears more than a certain proportion to his height. It is notorious that such persons bear even slight accidents badly, and succumb to illnesses that would be unattended with danger in healthy subjects. After death their tissues are found to be soft and flaccid, and to break down under pressure much more readily than usual, and decomposition often advances with undue rapidity. The omentum, the mesentery, and the subperitoneal tissue generally, are loaded with fat. The large size of the abdomen in fact presses up the diaphragm during life, and hampers the play of the lungs. The heart also is commonly covered with fat, and its substance is soft and lacerable, so that it tears readily and has been compared to wet brown paper.

The most important part of the treatment of obesity consists in the regulation of the diet. Some years ago popular attention was strongly drawn to this subject by a pamphlet published by Mr Banting, who, in less than a year had had his weight reduced from 202 to 156 pounds. He was at that time sixty-six years of age, and his height was five feet five inches. Before he began to diet himself he had great difficulty in stooping, was compelled to go downstairs slowly backwards, and used to puff and blow with every exertion. He was liable to fainting-fits. The articles which he specially avoided were bread, butter, milk, sugar, beer, sweet wines, potatoes, &c. He took a fairly liberal supply of animal food. He says that when he had lost his excess of fat he felt better than he had done for twenty years, and the fainting-fits altogether ceased.

Such a change of diet should not be made without supervision on the part of a medical man, for in some persons it may doubtless be attended with risks of its own. But the dangers which obesity brings with it far outweigh them. Habits of early rising and of active exercise may be useful in preventing the deposition of fat, but the latter is beyond the power of those who are already corpulent. Liquor potassæ and other alkaline remedies have been recommended in the treatment of this condition by some writers, but they often prove altogether useless.

4. *The lardaceous or waxy liver.*—I shall have occasion elsewhere to speak of the causes which lead to the conversion of the tissues of certain organs and parts of the body into a peculiar translucent material, which is known by the epithets lardaceous, waxy, &c. At present I merely wish to point out the characters that distinguish this from other forms of enlargement of the liver, which likewise give rise neither to pain, nor (as a rule) to ascites. A lardaceous liver often reaches a considerable size. I have notes of a case in which it weighed more than eight pounds, and Dr Wilks speaks of one in

which it weighed fourteen pounds. It often reaches down to the level of the umbilicus. During life the edge can generally be felt very distinctly, and is more readily discovered than that of a fatty liver. It is firm and resisting. The surface of the organ is perfectly smooth, unless perihepatitis or some other disease be also present. But such a combination is sufficiently common to need to be borne in mind, particularly as we shall see hereafter in reference to the diagnosis of hydatid tumours of the liver.

One of the most striking characters of a lardaceous liver in the post-mortem room is its greatly increased density. Wilks mentions an instance in which the specific gravity was found to be 1084. It is also extremely hard. Its cut surface looks dry, smooth, shining, and semitranslucent. It can be cut into thin slices much more readily than in health. The lobular markings are unduly distinct.

The microscope shows that the hepatic cells themselves are converted into lustrous shapeless masses of the lardaceous material. The cells earliest affected are those which lie at about the middle of each lobule. This position corresponds with the distribution of the ultimate branches of the hepatic artery. After a time the change extends inwards to the centre of the lobule, and last of all outwards to its periphery.

Dr Duckworth has recorded cases in which the lardaceous liver became reduced in size. From reaching to the iliac fossa and nearly to the pubes, in one case it became in fifteen months about half the size it was, and at the autopsy weighed only 117 ounces ('St. Barth. Hosp. Rep.,' vol. x, p. 57, 1874).

5. *Hydatid tumour of the liver.*—In all the forms of painless enlargement of the liver that have hitherto been mentioned the organ has been *uniformly* increased in size. In this respect they differ altogether from hydatid tumour, which is a rounded elastic swelling, occupying only part of the liver, although it may reach a very considerable size before its presence is detected. It often causes not the slightest inconvenience or discomfort to the patient, who remains in perfect health.

It has been stated in a former chapter (p. 215) that every tapeworm, in the course of its development, passes through a very remarkable phase, in which it forms a bladder, embedded in the substance of one of the higher animals, and filled with a transparent fluid. Now, a hydatid of the liver really represents a stage in the development of a little tapeworm, which in its mature form inhabits the intestine of the dog, and is called the Tænia echinococcus. When fully grown this measures at most four millimetres in length, and consists of only three or four segments, of which the last alone contains developed sexual organs. It is very common in London dogs and is often present in enormous numbers in their intestines. Its ova are discharged with their fæces, and in some way obtain an entrance into the human stomach, being probably carried there either in drinking-water or upon the leaves and stems of raw vegetables. When an ovum of the Tænia echinococcus has thus found its way into the alimentary canal of a man who is henceforth to be its "host," it at once enters upon a wonderful career of development. The first change appears to be due to the action of the gastric juice which dissolves its shell, and liberates the embryo contained in it. This embryo possesses six little hooks, arranged in two rows, and being capable of active movement it probably at once sets to work and bores its way through the walls of the stomach or of the small intestine, if it had passed through the pylorus with the chyme. Its further course is somewhat uncertain. Sometimes it appears to reach the serous surface and may either remain and develop itself within the peritoneal cavity, or perhaps strike across into one of the solid viscera. If, however, this were its usual course the hydatid which grows out of it ought to be met with in the other abdominal viscera almost as frequently as in the liver. But the fact is that hydatids are very much more commonly found in the liver than anywhere else. It can hardly be doubted that the cause of this is that the embryo, in piercing the wall of the stomach or intestine, generally gets into one of the rootlets of the portal vein, and is at once washed away by the stream of blood, and carried onwards through the main trunk, until at last it is arrested in one of the capillary branches of the portal vein within the liver.

Having thus reached the interior of the liver the embryo proceeds to develop itself into a hydatid. Perhaps it first bores its way out of the blood-vessel, and may even travel some distance through the hepatic tissue, as the embryos of some other tænia are known to do in the organs which they infest. Very soon, however, its movements are arrested. It loses its hooks, grows larger, and from being solid, becomes converted into a vesicle, containing a transparent fluid. It also gives rise to certain changes in the tissues round it, apparently as a result of the irritation caused by its presence. It becomes surrounded by a layer of granular matter, and before long by a distinct membranous investment, consisting of connective tissue, and abundantly supplied with vessels. Henceforth the hydatid is always enclosed in this investment, which grows as it grows, and which may be properly termed its capsule. Thus the capsule of a hydatid is really a structure formed from the human tissues, and the hydatid or echinococcus itself simply lies in contact with its inner surface, and possesses no organic connection with it.

Up to this point the development of the echinococcus is precisely analogous to that of the cystic stage of any other tænia, for instance, of a cysticercus. But the further steps are very different in the two creatures. The cysticercus, in order to complete its development, would only have to form a single "head" or "scolex" in its interior. This head would grow as a kind of bud or protrusion from one part of the interior of the cysticercus, and would gradually become provided with its two rows of hooklets and its suckers. Now, in most specimens of the echinococcus, instead of a single bud or protrusion, a number of them form at different times from the interior of the animal. And each of these buds does not develop into a head, but itself forms a cystic body which for a time remains attached to the spot where it arose by a pedicle, but soon becomes detached. It is then called a "daughter-cyst," while the original hydatid that encloses it is termed the "mother-cyst." Each daughter-cyst, again, may develop one or more "granddaughter" cysts in its interior. In this way the echinococcus becomes filled with a number of smaller vesicles of various sizes, which may amount to thousands. If any of them contain other smaller vesicles they are sometimes described as "pillbox hydatids," since their arrangement may be said to resemble that of a "nest" of pill-boxes. Sooner or later, the little buds or protrusions cease to form detached vesicles, and develop into very small thin membranous sacs, the pedicles of which are persistent, and which are called "brood capsules," because they give origin to a variable number of "scolices" or "heads," each of which has its row of hooklets and its four suckers, and is capable, under favourable circumstances, of growing into a tænia. These scolices or heads have in England been commonly designated echinococci; and the use of this term accords both with its derivation (ἐχῖνος, hedgehog, κόκκος, grain or berry) and with the intention of Rudolphi, who invented it. But at the present day, German writers apply the name of echinococcus (as I have done) to the whole animal with its daughter-cysts, brood capsules, and scolices. And this is certainly more convenient, because it corresponds with that adopted in the case of other round-worms. It will be observed that the difference between a cysticercus and an echinococcus is that, whereas the former gives rise only to one scolex, and can ultimately form only a single tapeworm, the latter may develop thousands of each.

Hydatids do not, however, necessarily pass through all the developmental changes that I have been describing. Sometimes they fail altogether to produce scolices, and even daughter-cysts. They are then said to be "sterile;" or they may be called "acephalocysts." The term acephalocyst was invented by Laennec, because he believed that scolices were never produced by the hydatid which infests the human subject, whereas he was aware of their presence in hydatids from the lower animals. It is said that Bremser, in 1821, first discovered scolices in hydatids taken from the human body. Bright was perhaps the earliest English physician to observe them; he gave a drawing of them in the 'Guy's Hospital Reports' for 1837. However, even after it was universally recognised that the hydatids of man contain scolices, they still continued to be called acephalocysts; and the term has never fallen entirely into disuse, although in this sense it is obviously inappropriate, whereas it might fairly be applied (as has been suggested above) to those hydatids which are really sterile. This is said to be more frequent in the case of hydatids infesting the brain than in those of any other organ.

The account given above does not, however, exhaust the list of developmental changes of which a hydatid is capable. Sometimes, but in the human subject very rarely, instead of budding internally to form daughter vesicles, it throws off protrusions externally. In this way the liver may become riddled with hydatids, not contained in any mother cyst, but penetrating its tissue in all directions, and even invading the neighbouring organs. A very remarkable case of this kind occurred a few years ago in Guy's Hospital, under the care of Dr Rees. A boy was admitted with what seemed to be effusion of fluid into the right pleura, and enlargement of the liver. But when the chest was punctured with a trocar, hydatids escaped. Ultimately he died, and it was found that the liver, diaphragm, and right lung were full of hydatids, which were budding externally in all directions. It has been supposed by some writers that when two or more echinococci are found in different parts of the liver, or in different organs of the body, the one has been derived from the other by the process of germination. But it is more probable that in such cases each hydatid was separately developed from a single embryo, the patient having swallowed more than one of the ova of the Tænia echinococcus.

Lastly, brief reference must be made to a very remarkable form of hydatid—developed by this process of external germination—which is styled "multilocular" by continental pathologists. This forms a solid globular mass in the liver, as large as a fist or a child's head. Its periphery is well defined, and it can be shelled out of the tissue in which it lies. But on section it is found to be divided by trabeculæ into a number of small cavities of irregular form, each containing a mass of gelatinous material which is made up of hydatid membranes pressed closely together, and small cysts containing scolices. The individual cysts are never larger than peas, and are often as small as millet-seeds. Virchow has suggested that in this form of the affection the parasite occupies the interior of the lymphatic vessels. This point, however, is at present doubtful. In its centre a multilocular hydatid tumour is always found to have undergone softening, and to be broken down into a suppurating cavity. Suppurative peritonitis and jaundice are also frequently present at the time of the patient's death. I am not aware that this form of hydatid tumour has hitherto been observed in England. Frerichs suggests that a specimen in the museum of Guy's Hospital which is labelled "colloid cancer of the liver" is perhaps really a multilocular hydatid. But some years ago I carefully examined this specimen, and could not discover any trace of a parasite in it.

We must now return to consider the characters of an ordinary hydatid tumour or echinococcus of the liver. This forms a more or less globular mass, varying in size from that of a walnut to that of a cocoa-nut, or even larger; the largest of which I have found mention is, I think, one weighing thirty pounds which was observed by Luschka. If it is subjected to no pressure in its growth its form is probably always spherical, but if it meets with more resistance on one side than on another, it may be flattened, or egg-shaped, or it may even assume an hour-glass form. The extent to which a hydatid is embedded in the liver varies greatly in different cases, and gives them very different clinical features. In some cases, it would seem that the six-hooked embryo originally lay just beneath the serous covering of the liver; and the hydatid may then form a globular mass, depending from its surface and having so little obvious connection with it that one may find great difficulty in determining that the liver is really the seat of the tumour. In other cases, a great part of the sphere formed by the hydatid may lie within the hepatic substance; and its presence may be indicated only by a rounded projection from one face of the organ, the curve of which is often little noticeable in proportion as the cyst is large. Sometimes, again, a hydatid may reach both surfaces of the liver at once; and the original anterior edge of the liver may then be distinctly made out as a narrow ridge, passing obliquely downwards and to the right, across the rounded tumour which occupies the epigastric and hypochondriac regions. Sometimes, lastly, a hydatid may be embedded entirely in the back part of the liver, or reach only that portion of its surface which is in contact with the diaphragm, and covered by the ribs.

Symptoms.—The sensations imparted to the physician by manipulation of hydatid tumours of the liver through the abdominal walls vary much in different cases. The tumour may be quite soft, and fluctuation may readily be detected in it, a wave being transmitted from one part of it to another, or even, if the echinococcus should be large enough, from the right hypochondrium to the right lumbar region, or it may be firm and tense, even sometimes of apparently stony hardness. In some cases a peculiar sensation may be elicited by percussion over it, to which Briançon first drew attention, and on which French writers generally lay great stress. It is termed the *frémissement hydatique*. The way to detect it is to place three fingers of the left hand upon the tumour, and then to tap the middle finger abruptly with the right forefinger. The other fingers of the left hand may then perceive a peculiar quivering sensation, which was formerly supposed to be due to the vibration of the daughter-cyst contained in the hydatid, but which (it is now known) may occur with cysts in which there is nothing but fluid. I cannot say that I have myself been accustomed to attach much importance to this sign.

Diagnosis.—As a rule, the diagnosis of a hydatid tumour of the liver, lying below the ribs, is not difficult. If the echinococcus should project far from the lower surface of the organ, it may be mistaken for a distended gall-bladder; and sometimes there may be a doubt whether the case is not one of hydronephrosis. Distension of the gall-bladder, without jaundice—the common bile-duct being patent—is, however, excedingly rare; and in hydronephrosis the tumour fills the lumbar region to an extent which can be very rarely the case with a hydatid; and, again, the colon is generally to be felt running over it.

A tumour which is distinctly cystic, and which at the same time is distinctly embedded in the substance of the liver, can, I believe, be nothing but a hydatid. Simple cysts of the liver appear never to reach such a size as would enable them to be detected during life, and are of merely pathological interest.

But when a hydatid is deeply embedded in the substance of the liver, so that it is but little raised above the surface of the organ, and yet causes it to project a long way down into the abdomen, there is often great difficulty in determining whether the case is really of this nature or not. I have known several instances in which the diagnosis was given that a tumour was a hydatid, but in which the enlargement proved to be due to lardaceous disease of the liver, and the nodulation to its being intersected by fibroid bands, in connection with syphilitic gummata. One diagnostic character of this affection of the liver is immobility of the organ during inspiration, due to adhesions of its surface which are generally present. More or less pain and tenderness on pressure are also common symptoms in such cases. It once happened to me to direct the performance of exploratory operations in two cases on the same day, in each of which the tumour proved to be solid; and I believe that in each it was a syphilitic and lardaceous liver. These patients did well; but I remember the case of another man who died of the effects of chloroform while undergoing an operation for a supposed hydatid of the liver, and in whom the tumour was found on post-mortem examination to be a solid mass of the nature just described.

In other cases there may be a difficulty in determining whether a tumour of the liver is a hydatid or a cancerous growth. The distinction must be based partly on the physical character of the tumour, partly on the presence or absence of symptoms of constitutional disturbance, particularly pain. It must, however, be mentioned that pain is not invariably absent in hydatid disease of the liver. Frerichs gives a case in which such a tumour was the seat of violent pains after every manipulation and movement, which pains ceased almost immediately upon the removal of a clear watery fluid by tapping. Again, we shall presently see that the capsule of a hydatid tumour of the liver may inflame and suppurate; and it then gives rise to very great pain. The health of patients infested with this parasite often appears to be perfect, but they may lose flesh to a certain extent; they may also suffer much inconvenience from the presence of the tumour in neighbouring organs.

But, as has already been stated, an echinococcus growing in the liver, instead of forming a tumour that can be felt in the abdomen, may project only from the convex surface of the organ, under cover of the ribs. If it should attain a considerable size it may then cause the lower part of the chest to bulge considerably, and the edges of the costal cartilages to form a much more open curve than on the opposite side of the body. The intercostal spaces over the swelling may feel more resistant than usual, and may even project beyond the level of the ribs. At the same time the lower part of the chest yields a dull note on percussion, and the case is very likely to be mistaken for one of chronic effusion of fluid into the right pleural cavity. Such an error may, however, be always avoided by careful observation of the limits within which the dulness on percussion and the enlargement of the intercostal spaces can alone be detected. In cases of subdiaphragmatic hydatid tumour the area of dulness is bounded above by a curved line, which descends as it approaches the spine posteriorly. In cases of pleuritic effusion the dulness reaches to quite as high a level in the dorsal region, close to the spine, as in the neighbourhood of the right nipple. A collection of fluid in the right pleural sac might, indeed, be so confined by adhesions as to be undistinguishable from a hydatid tumour. But this possibility need hardly be taken into account in a statement of the rule that where the physical signs indicate the presence of a collection of fluid limited to the lateral region of the base of the right chest, a hydatid tumour of the liver is present. Some time ago I saw a case with my friend Mr Durham, which admirably illustrates this principle. The patient, a young lady, had been sent to him by a physician who considered her to be suffering from a chronic pleuritic effusion, the result of an attack of pleurisy two or three months before. The right lower ribs, in the lateral region of the chest, were bulging, and the intercostal spaces were tense, and seemed to yield a sensation of fluctuation. There was increased dulness on percussion over the same part; but in the back the physical signs were in all respects normal. In spite of the history that was given us, Mr Durham and I formed the conclusion that the case was one of echinococcus in the liver; and the aspirator at once proved this conclusion to be correct.

For whatever doubt there may be as to the nature of a cystic tumour of the liver, it is quickly set at rest by the chemical examination of the liquid removed from it by paracentesis with a trocar or with an aspirator.

This fluid possesses characters different from those of any other liquid that is ever met with in the chest or abdomen, although they are not unlike those which belong to the cerebro-spinal fluid. Hydatid fluid is limpid or very slightly opalescent, its sp. gr. is 1007 to 1009, or (some say) 1013, there is no albumen in it, so that it does not coagulate either when boiled or on the addition of nitric acid. It is said, however, that it always contains a minute proportion of grape sugar, and succinate of ammonia has also been found in it. When a glass containing hydatid fluid is held up to the light, one can often see floating in it delicate white bodies, so minute as to be only just visible, which rapidly settle to the bottom of the vessel. These are the clusters of scolices, either still enclosed in their brood capsules or (if the latter are ruptured) kept together by their common stalk. They form beautiful objects for the microscope, appearing as bodies of round or slightly elliptical form, with oval calcareous corpuscles scattered through their transparent substance, and with their crown of hooklets and their four suckers usually retracted into their interior. Very often they are still alive, and can be seen to perform active movements. It was formerly supposed that the scolices, or echinococcus-heads, became detached from the main wall of the parent cyst in the course of their growth, and even that they possessed cilia and could swim about in the fluid. But this was a mistake; they are naturally fixed, and are set free during the operation of paracentesis.

The discovery of a hydatid scolex, or of one of the hooklets, whether in the fluid or the solid contents of a cyst of doubtful nature, is of course conclusive, and the membranous wall of a hydatid cyst also possesses microscopical characters which are entirely different from those of any tissue of the human body. This is the case, at least, with the outer of the two coats of a hydatid, or as it is technically termed, the "cuticula." It is made up of a number of very thin layers arranged concentrically. The smallest portion of it is seen under the microscope to be marked with delicate parallel lines, having a peculiar finely dotted appearance, which is perfectly characteristic. Another peculiarity which belongs to hydatid membranes is the fact that when they are lacerated, the free edge always rolls itself up so that the originally inner surface is outermost. Chemically they consist of a modification of chitin.

Results.—A hydatid tumour of the liver may give rise to a variety of consequences, some of which result in the cure of the disease. It is an interesting question whether there is any natural limit to the life of the creature. To this question no certain answer can perhaps as yet be given, although Reynal is said to have met with an instance in which a tumour of the neck which had existed forty-three years—from the age of seventeen to sixty—when punctured gave issue to an immense quantity of hydatids, all round and apparently living; and Dr Budd recorded the case of a lady who died at the age of seventy-three, and who was believed to have had two hydatid tumours since she was eight years old. It is at any rate certain that in persons who have died at a much earlier age than this, dead hydatids are frequently discovered. Very often their contents are deeply stained with bile. Cruveilhier long ago suggested that the entrance of bile into the capsule by ulceration of some small bile-duct has in such cases been the cause of the death of the parasite, and this opinion has since been very generally adopted. But it seems to me very doubtful, for I have more than once found two dead hydatids in the same liver, one of them containing bile-stained matters, while the contents of the other were colourless. It seems unreasonable to attribute the death of the one to the toxic action of the bile, and to leave the death of the latter unexplained; and I am disposed to think that the entrance of bile is really accidental, and that it occurs during the changes which lead to the shrinking of the tumour after the parasite has ceased to live. Another supposition has been that hydatids die because their external capsule is too thick and resistant to allow of their due growth. The capsule of a dead hydatid is often found to be of cartilaginous hardness, or even in great part calcified, and it is asserted that the mother-cyst (although it may be full of closely-packed daughter-cysts) does not appear to be folded, as though it had ever been of larger size. It is therefore imagined that the death of the hydatid was due to its being prevented from receiving the proper amount of nutriment for the supply of its multiplying daughter-cysts. But I have, in one instance of this kind (in which I paid attention to this point), found that the mother-cyst was much folded.

When a dead echinococcus is found in the liver at an autopsy, its capsule is generally found to contain a putty-like substance, made up in large part of calcareous salts, and mixed with the gelatinous relics of hydatid membranes, which often glisten with cholesterine crystals. The putty-like substance is very like that which occurs in a dried-up abscess, and in all probability it does in reality correspond to pus which had formed within the capsule of the hydatid, coincidentally with the death of the parasite or as a consequence of it.

Suppuration within the capsule of an echinococcus is, indeed, a not uncommon occurrence, and one which is very important, since it modifies greatly both the physical signs of the affection and its symptoms. If a tumour can be seen or felt in the abdomen this may become painful and tender, it may be felt to be hot, and there may even be redness of the skin over it. The patient's health begins to suffer; he may have repeated attacks of shivering, and symptoms of hectic may show themselves. In some cases, however, inflammation of the capsule of a hydatid may apparently take place without any very marked symptoms.

A suppurating hydatid may ulcerate and discharge in various directions, and, on the other hand, a living echinococcus may also burst its capsule and pour out its contents. In the latter case suppuration of the cavity follows, and thus, unless the death of the patient should follow directly, one can very seldom determine whether the creature was alive or not at the time when its capsule gave way. Writers have, in fact, hitherto made no attempt to distinguish between these two conditions.

One direction in which a hydatid cyst sometimes (but rarely) makes its way is through the abdominal parietes. It is said that this may happen even to a cyst in which the parasite is still alive, the discharge being then clear water. Another direction in which rupture may take place is into the peritoneal cavity, and this is often not a spontaneous occurrence but the result of some injury to the abdomen, such as the patient's falling downstairs or receiving a severe kick or blow, Fatal peritonitis generally follows quickly upon an accident of this kind, but unless it is known that the patient had a hydatid tumour previously, it must necessarily be impossible during his life to say why the injury should have given rise to such severe symptoms. At least two cases, however, have been recorded in which rupture of a hydatid cyst into the peritoneal cavity seems to have taken place without the patient having been much the worse for it, although there was for some time afterwards fluctuation in the lower part of the abdomen, just as in ordinary ascites. In all probability, I think, the different results of rupture in different cases depend upon the circumstance that sometimes a large number of daughter-cysts and scolices are effused into the peritoneal cavity (particularly when the tumour is widely ruptured by extreme violence), but that in other instances only the hydatid fluid enters the serous sac, this resulting either from the hydatid cyst being sterile, or from the aperture being small and the escape of the contents of the tumour slow and gradual.

Much more commonly, a hydatid cyst discharges its contents either into the stomach, or into some part of the intestinal canal. The daughter-cysts are then vomited or discharged per anum; and sometimes air enters the tumour, which thus becomes tympanitic on percussion. The evacuation of membranous portions of hydatids in the fæces sometimes goes on for several weeks or longer; and in the great majority of cases the patient ultimately recovers.

The rupture of a hydatid cyst into one of the biliary passages has already been referred to as one of the causes of febrile jaundice (p. 270).

The hydatid cysts which discharge their contents in any of the directions that have been hitherto considered are generally connected with some part of the liver which is within reach of the ordinary methods of physical examination. But sometimes the tumour bursts not downwards, but upwards, into the chest; and in these cases, its seat is almost always in the upper and posterior part of the liver, so that very frequently no positive physical signs of its presence can be discovered either before or after its rupture.

Sometimes, for instance, a hydatid cyst discharges its contents through the diaphragm into the pericardial sac; or, again, into one of the hepatic veins within the liver, the daughter-cysts in the latter case passing straight into the right chambers of the heart, and plugging up the branches of the pulmonary artery. The clinical features in both cases are very similar, consisting in the occurrence of sudden death, or, at least, rapidly fatal syncope, in a person who perhaps has hitherto appeared to be in perfect health.

In rare instances, again, a hydatid tumour has been known to discharge its contents into one of the pleural cavities (generally the right) with the result of setting up a severe and rapidly fatal pleurisy. But in the very great majority of cases in which the diaphragm is pierced by a hydatid, the pleura has become adherent before perforation takes place. The consequence is that the parasite makes its way into the substance of the lung, and sooner or later reaches a bronchial tube, into which it opens, so that its contents are expectorated. Cases of this kind are exceedingly interesting, and it is often a long time before their real nature can be made out.

Some years ago I saw a boy, aged six, who was a patient of Mr Fagge, of Hythe, and who had for about eighteen months had a pain in the lower part of the right chest, just outside the nipple, and a constant hacking cough, for which all treatment proved useless. He took to his bed, became exceedingly wasted, and was supposed to be sinking. One day his cough left him, and he became exceedingly prostrate, but next morning the cough returned, and he spat up a hydatid cyst and a quantity of pus. From that time he began to recover, and his cough gradually disappeared. Ultimately he died of another disease, and it was proved that the hydatid had originally come from the liver.

A young woman was once attending among my out-patients who had been expectorating hydatids for nearly a year when she first came to me. Next week she had a most violent attack of coughing, which lasted three hours, and in which it was feared that she would be choked. She at last got rid of a large piece of hydatid membrane, which was apparently a part or the whole of the mother-cyst; for she coughed up no more hydatids, and to a great extent regained her health.

When portions of hydatid membrane from the liver are expectorated they are generally colourless, but sometimes are deeply stained with bile. In many instances the patient regains his former state of health when all the hydatids have been voided, and the capsule has contracted so as to close the cavity. This process, however, is itself not entirely free from risk. I once made an autopsy in the case of a patient of Dr Habershon's who had spat up hydatids some months before, and in whom hæmoptysis came on which could not be arrested, and which before long proved fatal. It was found that the blood came from a branch of the pulmonary vein; this had become obstructed and dilated into a cylindrical tube as large as a lead pencil, and had afterwards opened into the cavity that had before lodged the hydatid, although this cavity was now shrinking. Here the hydatid had originally been seated not in the liver, but in the lung itself. When a patient goes on expectorating hydatids for several months, and in considerable numbers, the question is sometimes raised whether the parasite can be alive during this period, and be constantly forming fresh daughter-cysts. I believe, however, that this is not possible, except perhaps when the creature is of that variety which buds externally. But, so far as I know, when this is the case, the parasite traverses the lung in such a way as to avoid the bronchial tubes, so that none of the vesicles are expectorated.

Prophylaxis.—Before passing on to consider what is the proper curative treatment of hydatid tumours, I must say something as to the measures that can be adopted to prevent the echinococcus from entering the human body. Now, in London hydatids are present in the liver with tolerable frequency, but in Edinburgh this affection is exceedingly rare. In the United States, again, it is said to be very uncommon. On the other hand, in Australia it is greatly more frequent than in England, and among the inhabitants of Iceland it causes one seventh of the total mortality. In our own country, it is not likely that people will ever take precautionary measures against infection with hydatids beyond taking care when they eat raw vegetables to have them thoroughly cleansed. But it is very different in Iceland. There prophylaxis against hydatid disease is probably the most important of all sanitary questions. Now, according to our present knowledge, it is evident that this parasite would soon become extinct in all civilised countries if its cystic form were not liable to infest some other animal than man. Dogs acquire the tænia echinococcus only by eating the flesh of some creature in which the scolex is embedded, and they can very rarely have the opportunity of deriving this from the dead human body. Sheep and pigs are believed to be the chief animals besides man which harbour hydatids. Consequently, dogs should be prevented eating the offal of these animals, and should be excluded from all slaughter-houses. It has also been suggested that a purge should be periodically administered to each dog, and that the floor of every kennel should be frequently scalded with boiling water so as to destroy any ova of the tænia echinococcus that may have been voided with the evacuations of the animal.

Curative treatment of hydatid tumours is believed at the present day to belong to surgery rather than to medicine. At one time it was thought that the internal administration of iodide of potassium, or even of chloride of sodium, would sometimes lead to the disappearance of a hydatid cyst; and, on the other hand, it was supposed that the operation of paracentesis was attended with great risk to the patient. Neither of these opinions is now accepted, and I think that most observers are agreed that an operation should at once be performed whenever a hydatid is detected which is above the size of a billiard ball, and which appears to be increasing in size.

The operative procedures that may be adopted for the cure of the affection may be placed under two heads: first, some of the fluid may be withdrawn either by a fine trocar or by the aspirator; secondly, one or two fine needles may be inserted into the cyst, and may either be left in it for ten minutes and then carefully taken out again, or they may be connected with a galvanic battery, the current from which is allowed to pass through the tumour for ten minutes before the needles are withdrawn.

The operation last mentioned, that of *electrolysis*, is one which has been practised several times for me at Guy's Hospital by Mr Durham, and we published in the 'Medico-Chirurgical Transactions' for 1871 the reports of eight cases, in seven of which (if not in all of them) this plan of treatment was completely successful, being followed in most instances by the total disappearance of the tumour. Two electro-gilt needles were used, which were introduced into the cyst at a distance of one or two inches from one another. Care was taken to observe that they had entered the same cavity and could be made to touch one another. They were then attached to wires connected with the negative pole of a galvanic battery of ten cells, while the positive pole was made to terminate in a moistened sponge placed over the surface of the tumour at a little distance from the points of entry of the needles. The current was allowed to pass for about ten minutes, after which the needles were withdrawn.

When we first made trial of this operation, Mr Durham and I supposed that, if it were successful, its success would be attributable to the decomposition of the saline liquid contained in the cyst, by which it might be presumed that the hydatid would be killed. But further experience suggested a doubt as to the correctness of this opinion. In several of our cases fluctuation could be detected in the lower part of the abdominal cavity a few hours after electrolysis. In two instances a rash showed itself on the first or second day afterwards, resembling urticaria or scarlatina. Now, Dr McGillivray has recorded a case in which urticaria followed the discharge of fluid from a hydatid cyst into the peritoneal sac. It therefore seemed to us probable that the operation of electrolysis led to a similar escape of some of the fluid, and that this might really be the cause of the death of the parasite. We inferred that the same result would perhaps follow the introduction of the needles without any galvanic current (*acupuncture*), and I have since seen two cases in which this plan was adopted, with results which were perfectly successful so far as I had an opportunity of watching them.

The other operation, that of *paracentesis*, has been especially advocated by Murchison, and of late several observers have recommended that it should be done with the aspirator of Dieulafoy. The value of this last suggestion seems to me to depend entirely upon the position of the hydatid cyst. If a large part of its circumference be outside the substance of the liver, the aspirator may with advantage be employed to remove its contents, which this instrument can rapidly extract. But if the cyst be almost entirely buried within the solid tissue of the liver, I think that the aspirator is likely to do harm. The withdrawal of a very small part of the fluid contained in a hydatid, by means of a very fine trocar is often sufficient to destroy the parasite and to set up a process which will ultimately lead to the disappearance of the tumour. To do more than this is generally useless, and to exert forcible suction by means of an aspirator upon a cyst surrounded on all sides by the resistant tissue of a solid organ must involve some risk of setting up inflammation.

For it is of great importance to observe that the uncertainty at present existing with regard to the choice of operations for hydatid tumours depends hardly at all upon differences in the risk attaching to these operations in themselves: *all* of them are now known to be capable of curing the complaint in most cases with safety to the patient. The point that remains doubtful is whether one operation is more likely than another to be followed by suppuration of the cyst, and the necessity for subsequent paracentesis.

It very often happens that some weeks or even months after tapping (or after electrolysis) the tumour is found to have regained its former size, or even to exceed it. This has generally been supposed to render necessary a repetition of the paracentesis, and when the liquid collects again, it is tapped a third time, and so on. Now, I believe that the liquid obtained by these secondary operations almost always differs from hydatid fluid in containing more or less albumen. The augmented size of the tumour is, in fact, due, not to the continued life of the parasite, but to the effusion of serum within its capsule, and after a second or third operation this serum generally contains leucocytes in greater or less numbers, and passes into pus. On the other hand, if the real cause of the enlargement of a hydatid tumour after an operation be recognised, and if further interference be carefully abstained from, the tumour sooner or later begins to decrease in size again, and after a time disappears. I would therefore lay it down as a rule that no second operation upon a hydatid cyst should be performed within twelve months, unless there be reason to fear that suppuration has been set up within the capsule.

With regard to the question whether there is less danger of the occurrence of suppuration after electrolysis (or acupuncture) on the one hand, or after paracentesis (with the fine trocar or with the aspirator) on the other hand, I believe we are not yet in a position to speak with certainty. Mr Durham and I have laid stress on the fact that in one series of eight (it might now be said ten) cases treated by the former method, there was not a single instance in which suppuration occurred, or in which any symptoms arose beyond those of very transient febrile disturbance. One patient was up and about the ward on the fourth day and was discharged from the hospital on the tenth day. I believe that no series of cases treated by paracentesis has as yet been published in which there has been a similar immunity from severe inflammation of the cyst. Dr Duffin has, however, remarked that children have formed the majority of the patients on whom the operation of electrolysis has hitherto been performed, and that this may perhaps be the reason why that operation has not hitherto been followed by suppuration. I know, however, from my own experience that hydatid cysts may suppurate, under other conditions, even in children. Dr Duffin is disposed to think that the presence of a larger number of daughter cysts in a hydatid tumour is a main cause predisposing to the development of suppurative inflammation in its capsule, but I am not sure about this.

Another point that must not be forgotten, in considering the relative results of paracentesis and electrolysis, is the possibility that the cyst may have undergone inflammation or even suppuration, without any very marked symptoms, before the operation. I am not sure that this really ever occurs, but it would certainly make electrolysis (or acupuncture) dangerous ; and therefore this operation should be reserved for cases in which the patient has been in perfect health, and has experienced not the slightest pain nor uneasiness in the tumour.

Even when a considerable time has elapsed after the apparent cure of a hydatid tumour there still remains a liability to the occurrence of suppuration within it. Dr Wilks has mentioned to me more than one instance in which this has happened after a long interval. The retrograde changes which lead to the ultimate disappearance of such tumours are no doubt very slow in their progress. A year or two ago, a patient of Dr Moxon's lay for several weeks in the clinical ward in bed, on account of a pain in the hepatic region which had come on some time after the performance of an operation for a hydatid tumour. The tumour itself could no longer be detected, but the pain was intense and resisted all the measures that we employed for its removal. Doubtless, some of the sensitive structures of the abdomen were subjected to traction by the shrinking of the capsule of the cyst.

Besides the operations that have been hitherto alluded to, several others have been proposed and advocated ; among them are the injection of oxgall, iodine, or oil of male fern into the hydatid cyst ; the introduction of a large trocar and the formation of a fistulous opening *ab initio*, the penetration of the tissues external to the tumour by gradual stages, so as to allow of the formation of adhesions between the capsule of the cyst and the parietal layer of the peritoneum. But when these various plans were proposed, it was not known how safely and successfully hydatid tumours may be treated by the simpler methods above described.

When suppuration has once occurred within the capsule of a hydatid tumour, the cyst should at once be tapped. In very rare cases, this cures the disease, and pus is not afterwards again formed, but almost always the operation has before long to be repeated. A large trocar should then be used, and if the tumour contains secondary cysts, as many of them as possible should be removed. Generally speaking, I believe it is wise now to establish a fistulous opening, and to have the cavity washed out every day with a weak solution of the permanganate of potass, or of carbolic acid, so as to reduce to a minimum the horrible fœtor which belongs to decomposing hydatid membranes. The obliteration of the cavity in such a case is necessarily a very slow process, and attended with much risk. But I believe that with proper management and good nursing, cases of this kind almost always terminate favourably.

Hydatids in other organs.—Although this is not strictly the proper place for it, I think it will be convenient for me to say here all that need be said with regard to the occurrence of the echinococcus in other parts of the body, instead of in the liver. The fact is that hydatids are comparatively seldom found anywhere else, and that they still more rarely have any clinical interest attached to them. Leuckart appears to think that for every three cases of echinococcus of the liver there may perhaps be one in some other organ, but I should be inclined to suppose that this estimate is above the mark. Davaine collected very carefully all published cases of hydatids occurring in various parts of the body, and he found that among 200 cases of this kind (those of the liver being excluded) there were about 40 cases of hydatids of the lungs, about 30 of the muscles and subcutaneous connective tissue, 30 of the kidneys, 26 of the pelvis, 20 of the nervous centres, 17 of the bones, and 10 of the heart.

When an echinococcus develops itself in the interior of one of the *lungs*, it is generally found in the base of the right lung, a circumstance which can be explained only on the supposition that the six-hooked embryo penetrated into the organ from the liver by its own movements. Clinically, hydatid disease of the lung is scarcely likely to be suspected until one or more of the daughter cysts have been expectorated, and even then it can very seldom be possible to distinguish a case of this kind from one in which the original seat of the parasite was the back part of the liver.

Hydatids of the *brain* have occasionally been met with in the post-mortem room; its symptoms are undistinguishable from those of other cerebral tumours. It may be remarked that the capsule of the parasite is exceedingly thin and delicate when the brain is the organ infested by it.

Echinococci are sometimes found in the *heart:* they give rise, according to their exact seat, to very varied symptoms or sometimes to none at all.

The *spleen* is but seldom the seat of a hydatid tumour. I remember one case of the kind in which there was a large tumour in the left hypochondrium, but in which, until an autopsy had been made, it remained uncertain in what organ the parasite had developed itself.

In some of the great cavities, such as the *pleura* and the *pericardium*, the echinococcus may grow to a considerable size, without any tendency to the formation of a capsule round it,—the natural serous membrane seeming to take its place. This is said not to be the case with the *peritoneum*, in which hydatids are described as always having a proper capsule. However, hydatid disease of the peritoneum has considerable clinical interest. One form of it is apt to be mistaken, even by the most skilful surgeon, for cystic disease of the ovaries; and an attempt has several times been made to remove such a tumour by ovariotomy. In another form of this affection a number of distinct globular tumours are found in different parts of the abdominal cavity, some of them having been developed in the omentum, and others in the interspaces between the different viscera. The physical characters of the tumours in such cases ought to render them by no means difficult of diagnosis.

Lastly, there is a very remarkable variety of hydatid tumour, which develops itself in the *pelvis*, between the bladder and the rectum, or (in the female) behind the uterus. In these cases the six-hooked embryo doubtless got into the serous cavity when it had penetrated the walls of the stomach, and fell by its own weight into the most depending part of the peritoneal sac. The result is the formation of a tumour which may assume an oval form exactly like that of a distended bladder, and may occupy precisely the same situation. I remember a case of this kind in which it was supposed that the bladder was full, but in which of course the catheter failed to give any relief. The patient died, but it was not until the case had been cleared up by an autopsy that its real nature was even suspected. Bright relates a similar instance; several others have been placed on record by different observers, and I think it may be laid down as a rule, that whenever a fluid tumour is felt in this position, which cannot be reduced in size by the introduction of a catheter into the bladder, one should think of the possibility that an echinococcus may be present. The hepatic region should be carefully examined in such cases, and indeed whenever there is reason to suspect the existence of hydatids in any other part of the body. For it happens very commonly indeed that the liver is at the same time infested with the parasite.

DISEASES OF THE SPLEEN AND OF THE LYMPH-GLANDS

Physical signs of an enlarged spleen—Abscess of the Spleen.
SPLENIC LEUCHÆMIA.—*History—Definition and characteristic symptoms of the spleen, the blood, and other lymphatic organs—Ætiology—Diagnosis—Pathology, course and treatment.*
HODGKIN'S DISEASE.—*Anæmia lymphatica—History—Ætiology—Morbid anatomy and pathology—Course, prognosis and treatment.*
TUBERCULOSIS OF THE LYMPH-GLANDS.

I have already spoken of the spleen as undergoing enlargement in certain specific diseases, especially ague (vol. i, p. 245), and enteric fever (vol. i, p. 188), and have also mentioned the occurrence of lesions in that organ, in ulcerative endocarditis, in cirrhosis of the liver, and under various other conditions. But there remain a very few affections in which the spleen is either the only part of the body to suffer, or at least may be regarded as the primary seat of disease; and these may be conveniently described in the present chapter, in which I shall also discuss such diseases of the lymphatic glands as are widely distributed throughout the body, inasmuch as they are closely connected with corresponding changes in the spleen—clinically as well as pathologically.

Physical diagnosis.—But first it may be well that I should enter into some details with regard to the determination of the position and size of the spleen by percussion, inasmuch as German observers have of late years defined much more accurately than before what can really be done in this direction. It had been pointed out by von Luschka that the long diameter of the organ inclines downwards and forwards parallel with the lower ribs on the left side, and that its normal extent corresponds very closely with the space from the ninth to the eleventh rib. But about one third of the upper and hinder part of it is covered by the inferior border of the lung, and is therefore inaccessible to percussion. Consequently, as Weil and others have shown, the arch of splenic dulness, as it can be mapped out upon the surface of the chest, forms a figure which is rounded in front and below, whereas above it is flattened; behind there is no definite limit to it, and it merges insensibly into the dulness produced by the kidney and other structures in the loin. Its length, in the line of the long diameter of the spleen itself, is usually about three inches; its breadth, in a direction at right angles with the length, is about two or two and a half inches; the distance of the lower and anterior extremity of the organ from the edge of the costal cartilages is about an inch and a half or two inches. I may add that the position of the patient at the time when percussion is being practised may either be that of sitting upright or that of lying towards the right side upon the right shoulder and the right hip, with the left side raised just sufficiently to make the lateral region of the chest accessible. It is also necessary to mention that the splenic dulness is always rather "superficial" or incomplete in consequence of the comparatively small thickness of the organ. Light percussion is therefore needed for its determination; and in some cases, especially when there is much subcutaneous fat, when the lung is emphysematous, or when the stomach and the colon are much distended, its extent cannot be accurately mapped out.

When the spleen becomes enlarged, its lower and anterior extremity projects more and more downwards and forwards, and soon can be felt below the costal cartilages. It then constitutes an "abdominal tumour," the seat and the shape of which will be described further on. It is said, however, that a spleen of normal size may be displaced, so as to lie far below its usual seat, and even to reach the left iliac fossa. Guttmann states that he has seen three cases of this kind. In one instance, that of a man aged thirty, the organ was put back and kept in position by a bandage, whereupon the pain from which he suffered, and which had been treated in various ways without success, immediately disappeared. In another patient, a woman aged forty-eight, the dislocation of the spleen began as the result of a violent physical effort; she felt it at the time. The third case is remarkable on account of the diagnosis having been verified by extirpation of the organ: recovery after the operation took place in a fortnight.

Abscess of the Spleen.—The wedge-shaped infarctus in the spleen which are formed by emboli in cases of heart disease or after fevers sometimes undergo suppuration and may then burst into the peritoneal space. But, from a clinical point of view, it is scarcely necessary to describe such an affection as an independent disease.

Instances however occur of severe and even fatal illness which is found at the autopsy to have been due to the formation in the spleen of a single abscess, for which no cause can be discovered, and which may reach a very considerable size. A case of this kind was recorded by Bright in the 'Guy's Hospital Reports' for 1838; about half of the substance of the spleen was involved, and there was an opening into the colon. He also gave another case, in which a sloughing abscess communicated with the stomach; but it is perhaps doubtful whether the original seat of the suppuration in this instance was the spleen itself. Some years ago I saw a similar case in the post-mortem room. The symptoms in every instance are obscure, consisting chiefly of pain in the upper part of the abdomen, frequent vomiting, rigors, pyrexia, and emaciation. A more or less distinct fulness or induration may also be discovered in the left hypochondrium.

During life I should think that it would be impossible to distinguish cases of this kind from others in which suppuration occurs outside the spleen, but immediately in contact with it, and which are probably much more frequent. In the 'Guy's Hospital Reports' for 1874, I recorded nine or ten such instances. In some of them there was a clear history of a blow or a fall having been the starting-point of the disease; in others a chronic ulcer of the stomach seemed to have set up inflammation outside that organ. One of the most remarkable among them was the case of a man, aged thirty-seven, who was admitted for a large rounded tumour in the left hypochondrium, reaching down nearly to the umbilicus, and displacing the heart upwards. He had been kicked in the left side nine weeks before. When he had been in the hospital for some time, it was found that the beats of the heart gave rise to splashing sounds in the tumour, of metallic or musical quality. He had previously vomited blood, and it was evident that there had been ulceration into the alimentary canal (probably into the stomach), and that the tumour now contained air as well as fluid. Yet the man's health appeared to be perfectly good, and he insisted on leaving the hospital about eleven weeks after his admission. Some time later he came back and said that the tumour had suddenly disappeared, having discharged a quantity of matter, which I think he had vomited, although the report does not positively state this to have been the fact. On examination no trace of the swelling could be discovered. Another very interesting case was that of a woman who was admitted eleven days after having crossed the Channel in a steamer on a very rough day: she had been very sea-sick, and had also been thrown out of her berth upon the cabin floor. A large tumour rapidly formed in the epigastrium, and in the left hypochondrium there was a second mass in the position of the spleen. It appeared not improbable that in the first instance blood was effused over the surface of the stomach and other organs, and that the process of suppuration was secondary. In three other cases a sub-diaphragmatic abscess perforated the pleura and set up a fatal empyema.

Again, I have met with one case in which I believe that I mistook for a splenic abscess one seated in the substance of the abdominal walls. In 1870 I was consulted by a gentleman, aged fifty-six, on account of a rounded, tender, and rather painful swelling, occupying a large part of the left side of a very protuberant abdomen. I had no hesitation in declaring it to be an enlarged spleen, and this opinion was confirmed by his pallid appearance, and by the fact that there was a decided excess of leucocytes in the blood, as many as forty being counted in a single field of the microscope. However, the skin over the centre of the tumour gradually became reddened, hot, and indurated, so that it could not be moved or pinched up, and at the end of a month Mr Durham made a puncture with a fine trocar, and drew off some pus. For some months afterwards there was a discharge of matter from the opening, but it ultimately closed, and the swelling entirely disappeared. Still no doubt was entertained that the case had been one of abscess of the spleen. But ten months later a second large tumour formed in the right side of the abdomen. This also was opened by Mr Durham, and a great quantity of very fœtid pus escaped. The patient was now exceedingly prostrate and ill, but he again recovered, and in 1876 I saw him in excellent health. On the whole, I think it is most probable that the real seat of the abscesses was between the layers of muscles in the walls of the abdomen.

Splenic Leuchæmia.—In 1845 Dr Hughes Bennett, of Edinburgh, recorded a fatal case of enlargement of the spleen in which, after death, the blood was found to be full of bodies which he regarded as pus-cells, attributing them to a "suppuration of the blood." A month later Virchow published a similar case, but he perceived that the abnormal blood-elements were identical with the colourless corpuscles that are now called leucocytes, and he proposed to call the affection "leukæmia." A contest for priority arose, in regard to which no decision need now be given, especially as modern pathology has deprived the rival views of most of their significance, by showing that pus-cells themselves are merely leucocytes. It was found that Donné had observed the microscopical characters of the blood in a case of Barth's in 1839, and that Velpean as far back as 1827 had recorded an instance in which the blood looked as if mixed with pus, there being also a great increase in the size of the spleen.* In 1846 Dr Fuller, of St George's Hospital, and Dr Walshe, of University College Hospital, demonstrated the change in the blood of living patients, and the disease has ever since been universally recognised. The name of leuchæmia is now generally adopted, for German writers unanimously refused to accept that of leucocythæmia, which was at one time commonly used in England, having been first proposed by Dr Bennett in 1851, when he gave up his original opinion that the blood contained pus. Either designation seems to me to be sufficiently accurate for all practical purposes, notwithstanding that the blood is never actually white, nor is it made up entirely of leucocytes, nor distinguished from healthy blood by the mere presence of these bodies, but only by their being unduly numerous.

The fact that the blood contains an excess of leucocytes is not, however, in itself sufficient to characterise a case as one of leuchæmia. Virchow long ago pointed out that they may be slightly increased in numbers under various conditions attended with irritation of lymphatic glands, as well as during pregnancy, and in fevers. He proposed to distinguish all such minor degrees of blood-change under the name of "leucocytosis"; and Magnus Huss has suggested that the line should be drawn at the point where the proportion of white cells to red discs reaches one in twenty, the presence of leuchæmia being admitted whenever this is the case. But, again, an extreme degree of leuchæmia may occur without any appreciable lesion of the spleen. This was pointed out by Virchow as far back as 1847; in the first volume of his 'Archiv.' he related an instance in which the principal morbid change was in the lymph-glands, which were enormously enlarged throughout the body. Subsequently he described two forms of leuchæmia —the one "splenic," the other "lymphatic"—distinguished by differences in the size of the leucocytes, these being comparatively large and having sometimes more than one nucleus in the splenic form, but in the lymphatic form being small, and having their scanty protoplasm in close contact with a solitary nucleus. This microscopical distinction is still admitted by Mosler and others. But subsequent experience has shown that to speak of a "lymphatic leuchæmia" as comparable with the splenic affection is attended with great practical inconvenience. Virchow himself soon became aware that the change in the blood was often absent when there was a general enlargement of the lymph-glands; and in the 'Krankhaften Geschwülste' he described under the heading of "lympho-sarcoma" cases of this kind, for which other German writers have used the singularly inappropriate name of "Pseudo-leukæmie." But when I come to speak of "Hodgkin's disease" I shall have to state that it appears impossible to draw any boundary line between cases attended with leuchæmia and those in which the proportion of white cells to red discs is nearly, or quite, normal. In fact it would almost seem as though the presence or absence of the blood-change were in that disease a matter of very little importance. But I must add that cases also now and then occur which can only be described as presenting a combination of splenic leuchæmia and of Hodgkin's, disease; a marked example of this, in which I made the autopsy, is recorded by Dr Frederick Taylor in vol. xxv, p. 246, of the 'Path. Trans.' Apart from such exceptional instances, splenic leuchæmia is distinguished from Hodgkin's disease by the three following characters:—(1) enlargement of the spleen is present from the commencement, it is enormous, it consists in a simple overgrowth of the splenic tissues, there being in the organ no scattered white nodules; (2) the occurrence of an excess of leucocytes in the blood is constant, and their numbers are very great; (3) an affection of lymphatic glands and of various other organs and tissues begins (if at all) much later than that of the spleen, it is comparatively slight in degree, it has scarcely any tendency to assume the form of definite tumour-masses.

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* Piorry had described a case as one of inflammation of the Blood, "Hémite."—Ed.]

(1) In the majority of cases the first thing noticed by the patient is that his abdomen is becoming larger, that there is a fulness or tumour in the left side, or that he has a dull aching pain there. On examination one generally finds that the spleen is already very large; even at this period its size is seldom less than that which would correspond with the most advanced stage, and with the most extreme degree, of any other disease. Sometimes, indeed, it does not reach below the umbilicus; but in many cases it descends to the level of the iliac crest. And as time progresses, it occupies a position which could hardly have been anticipated for it, and which I illustrated by a series of diagrams in the 'Guy's Hospital Reports' for 1869. The vessels at its hilus seem to offer a resistance to its expansion in a straight line, and it therefore becomes curved, its lower end sweeping across

the brim of the pelvis, and even turning upwards when it has reached the right iliac fossa. So completely may the organ fill the whole abdomen below the navel that in the female it has often been mistaken for an ovarian tumour. I should, however, add that Mr Spencer Wells speaks of it as sometimes projecting downwards behind the uterus, so as to be felt there through the wall of the vagina. In any case, its real nature may be easily distinguished by the sharp edge which crosses the abdomen obliquely from the left lower ribs downwards, and which presents one or more notches, generally clearly cut and deep in their outlines. Its surface is almost always smooth and firm. A friction-fremitus can sometimes be felt over it, and with the stethoscope a blowing systolic murmur is often to be heard. At an advanced stage of the disease it may be separated from the parietes by a layer of ascitic fluid, through which the fingers have to dip, before they reach it. A patient of mine who was still living in 1880 had been tapped two and a half years previously, when three and a half gallons were removed.

After death the spleen is often found to be fixed to the adjacent parts by numerous adhesions. Its capsule may present large white or yellowish-opaque patches of thickening. Its cut surface is generally smooth, shining, and homogeneous looking; but sometimes it is marked with whitish lines and striæ, due to thickenings of the trabeculæ. Its consistence is often very firm, and its colour may be brownish rather than purple. It not infrequently shows a number of wedge-shaped pale infarctus, resembling those which are seen as results of embolism. Histologically the only change discoverable is overgrowth of tissue elements exactly like those of which the organ consists in its normal state; but the stroma often has a peculiarly well-marked fibrous character.

(2) The blood in splenic leuchæmia is paler than natural, and may look slightly turbid; indeed, when the excess of leucocytes in it is very great, it sometimes has a greyish-red colour, resembling a mixture of pus with blood. Charcot's crystals have sometimes been found. After death the appearance of coagula in the heart, or in the great vessels, is often very peculiar; they are grumous, and opaque, and have been likened to solidified pus.

The proportion of leucocytes to red discs varies widely in different cases, and according to the stage of the disease. From the normal ratio, which is not higher than 1 to 300, it may be increased to 1 to 8, 1 to 3, 1 to 2. The leucocytes may even be the more numerous, as in a case of Sörensen's, in which they were counted, and found to be as 68 to 47 of red discs. The number of red discs, on the other hand, is always much diminished, the total number of corpuscles in a given volume of blood being constantly less than in health. It is remarkable that this anæmia, or spanæmia, is commonly unattended with very obvious pallor of countenance; as Wilks long ago pointed out, patients, even at an advanced stage of the disease, have often a good deal of colour in their cheeks and in their lips, so that, seeing them in bed, one would hardly imagine them to be very ill. But sometimes the skin is yellowish white, like wax; it was so in the only infant whom I have seen affected with splenic leuchæmia. Some years ago Mr Golding Bird, at my request, examined blood from a case of leuchæmia, upon a warm stage; many of the leucocytes were found to be in active movement, but others remained motionless. In 1880 Dr Cavafy read a paper at the Royal Medical and Chirurgical Society upon a case, already far advanced, in which he repeatedly made observations of this kind: the proportion of leucocytes which showed even slight amœboid movements was at first only 12 per cent., at a later period only 6 per cent. He concluded that the greater number of these were dead or dying, and incapable not only of development, but even of emigrating through the walls of the vessels.

Other observers have stated that in some cases of this disease many of the leucocytes are obviously in a state of fatty decay.

The blood-change is probably the cause of some of the symptoms of the disease which have yet to be mentioned. By diminishing the amount of oxygen which can be taken up, it may be in part concerned in producing the *dyspnœa*, which is sometimes the chief thing complained of by the patient. This may be present only when muscular efforts are being made; in extreme cases even the slightest movement is attended with the utmost distress. Perhaps, therefore, there is nothing surprising in an observation made by Pettenkofer and Voit,[*] according to which, during rest, the quantity of oxygen absorbed and that of carbonic acid given off seem the same as in health. But another way of accounting for the dyspnœa is to ascribe it to the displacement of the diaphragm upwards by the enlarged abdominal viscera.

The over-abundant leucocytes are probably very apt during life to adhere in large numbers to the lining membrane of small vessels, and may even accumulate so as to obstruct them after the manner of minute emboli. And if it be a fact that many of the cells no longer possess their vital properties and are actually undergoing disintegration, nothing is more likely than that they should set up a morbid change in the walls of the capillaries with which they come into contact, softening them, and rendering them liable to rupture. In this way one may explain a marked symptom of splenic leuchæmia, namely the tendency to *hæmorrhages*. Thus epistaxis is very common; it may recur every day, and it sometimes is the direct cause of death. Bleeding may also take place from the intestine, the stomach, the kidneys, the lungs, or the uterus. It must be added, however, that the hæmorrhage after a wound or other injury is apt to be very excessive; the extraction of a tooth even has, in one case, led to fatal consequences. The statement has indeed been made that the amount of fibrin yielded by the blood in this disease is above the normal, but that instead of coagulating in long elastic filaments, when separated by stirring, it falls in peculiar granular fragments. Purpuric spots are frequently seen upon the skin; and after death the surface of the heart may be found ecchymosed. A large quantity of blood may be extravasated among the muscles or behind the peritoneum. Lastly, hæmorrhage into the brain sometimes occurs, with the symptoms of apoplexy.

In the retinæ, again, hæmorrhages are frequently observed, both during life and after death. Dr Gowers, in an article on the subject in ‘Reynolds’ System of Medicine,’ says that they are usually small, and most abundant towards the periphery; they often form striæ, following the lines of the nerve-fibres. After a time the blood may undergo conversion into a brownish pigment. But another feature of the hæmorrhagic patches is that they may have white or yellowish-white spots in their centres. Similar spots may also be observed without any accompanying extravasations. Dr Gowers has even seen the retina affected with parenchymatous swelling, and its veins greatly distended and tortuous. Thus a “leuchæmic retinitis” may fairly be spoken of. The changes in question, however, have a clear resemblance to those which occur in Bright’s disease. Have observers always been sufficiently careful to exclude the possibility of its presence as a complication?

(3) Several of the organs and of the tissues are liable to changes in splenic leuchæmia, but very few of these changes can at present be said to have any clinical significance. In about one of every three cases there is enlargement of *lymph-glands*, especially those of the abdomen and of the chest. The increase in their size is considerable; in our records I find that it is often noted that they were twice as large as normal. They may be firm and fleshy or soft and medullary in character. They never, I

[*] ‘Zeitschft. f. Biol.,’ v, 319: 1869. The figures were: O 790 to 832, CO_2 265 to 249.—Ed.]

believe, become fused together, nor does a new growth ever start from them and penetrate into structures adjacent. The *follicles* at the base of the tongue and the *tonsils* may be greatly swollen, and here an inflammatory change may also be present, for in one case at Guy's the left tonsil was found after death to be sloughing. There may also be a diffused pharyngitis, and a stomatitis, which Mosler supposes to be consequent upon the perverted state of the buccal secretions. The gums may become swollen, ulcerated, or gangrenous. The *intestinal follicles,* solitary and agminated, are sometimes greatly enlarged, and a lymphoid growth is said to extend beyond the limits of the glands and to infiltrate the submucous tissue. Even ulcers may form, which are described as having thickened edges, and as resembling tubercular ulcers rather closely.

But among the most remarkable of all the changes in the natural lymphoid structures of the body are those which occur in the *medulla* of the bones, and which were first described by Neumann. They give to the cancellous tissue a greenish-yellow appearance, exactly like that which is seen in osteomyelitis, and on pressure a puriform juice exudes.

Mosler met with a case in which a leuchæmic affection of the sternum was indicated during life by great tenderness on pressure over it.

It is a curious circumstance that in some instances wedge-shaped infarctus exactly like those which are commonly seen in pyæmia, are found in the *lungs.* This was the case in a patient whose spleen was excised by Mr Bryant in 1866, and who only survived the operation three hours. The patches which occupied the back parts of the lungs had gangrenous centres and red borders. Dr Gowers speaks of such infarctus as arising from plugging of the pulmonary capillaries by leucocytes; but I think that even if this mechanical theory be adopted, the relation to pyæmia must be admitted to be very close. I am not aware that the lungs in splenic leuchæmia ever exhibit definite nodules of a new growth such as are seen in Hodgkin's disease. In many instances they are quite healthy.

Of the remaining organs, the *liver* is most frequently diseased in splenic leuchæmia. It not infrequently weighs as much as eight or ten pounds. There is not always any obvious change in the appearance of its cut surface, and Virchow has even stated that the enlargement may be due simply to over-growth of the hepatic cells. But in many instances there are masses of lymphoid growth scattered through the organ, especially in the neighbourhood of the vessels. Sometimes these are visible only with the aid of a lens, sometimes they are apparent as minute greyish-white granules. Moreover, even within the acini numerous leucocytes are seen between the hepatic cells; some appear to lie within the capillaries, others are outside them, being supported by a nucleated stroma of their own. It does not appear that these changes ever cause jaundice. The *kidneys* are less often affected; in them the lesion assumes the form of scattered greyish-white striæ, running through the cortex, and bearing a close resemblance to those which are seen in cases of ascending nephritis. The urine is not infrequently albuminous during life, but sometimes, though not always, this is to be accounted for by the presence of other changes in the kidneys, such as occur in ordinary Bright's disease.

(4) To complete my description of the symptoms of splenic leuchæmia I have still to mention that the patient may complain of weakness, depression of spirits, headache, giddiness, noises in the ears, palpitation of the heart. In many cases *pyrexia* is present from time to time, lasting a few days, and being followed by intervals during which the temperature is normal. According to Dr Gowers the thermometer generally ranges up to 100° or 101° with the usual daily fluctuations, but it may reach 102°, 103°, or even 105°. Sometimes the pyrexia is accompanied with shivering and sweating.

Ætiology.—In the majority of cases no *cause* for splenic leuchæmia can be discovered, but in a certain number it appears to be a remote sequela of *ague.* Some of the writers who first studied the disease thought that it bore no relation to the marsh poison because it often occurred in persons who had never been exposed to that poison, and because the affection of the spleen resulting from ague was then regarded as merely congestive. But I believe that whenever several cases of leuchæmia have been collected together, some of the patients have always suffered from intermittent fever. Dr Gowers says that this was so in thirty among 150 cases which he collected, and at Guy's Hospital I find that the proportion has been considerably higher. There is clearly something more than a mere coincidence in these results, but it is remarkable how long the interval has sometimes been, and how mild the attack of ague to which such serious consequences are traced. In nine out of twenty-one cases a period of from ten to thirty years passed before the leuchæmia showed itself by any symptoms. In 1880 I had a patient in Guy's Hospital, a Pole, who had had had tertian ague in 1858 during four months in Warsaw, but who first noticed an enlargement in the left hypochondrium in 1866. In women splenic leuchæmia seems often to bear a relation to the sexual processes; sometimes it is discovered during pregnancy, sometimes it seems to arise out of the debility resulting from parturition. Of other supposed exciting causes, such as over-fatigue, distress of mind, intestinal catarrh, blows upon the abdomen, one may perhaps say that their connection with the disease was probably only apparent.

It occurs about twice as often in males as in females. It affects persons of all ages, from infants to those who are over seventy years old, but it is most frequent in those who are between twenty and fifty.

The *diagnosis* of splenic leuchæmia offers difficulties only in the early stages, with the exception of certain cases in which the characteristic symptoms fail to develop themselves fully, or which appear to be transitional between it and Hodgkin's disease. Mosler says that it is apt to be taken, in the first instance, for a mere chlorosis. But I believe that if a large splenic tumour and a great excess of leucocytes in the blood are at any time present, well-marked indications of these changes may always be discovered before there is pallor. It is otherwise with those cases in which the spleen remains moderately increased in size, and in which the proportion of leucocytes to red discs is but slightly augmented, while the anæmia is perhaps extreme. We have had more than one case the true nature of which has been left doubtful even after the autopsy.

There may be great enlargement of the spleen without any leuchæmia. Dr Moxon cites two such instances, one of which occurred to Mr Spencer Wells, the spleen weighing 6 lbs., the other to Mr Squire, the spleen weighing 13 lbs. Dr Gowers, following Griesinger, terms this affection "splenic anæmia." I have never met with an example of it. Again, there may be an excess of white corpuscles in the blood, while the spleen remains of natural dimensions, or nearly so. I think that if the excess is very great—as in a case related by Dr Goodhart, in the 'Clinical Society's Transactions' for 1877, although the spleen weighed only seventeen ounces after death, one may set down the disease as splenic leuchæmia. There is as yet no proof that a primary lesion of the spleen can lead to an excess of white corpuscles in the blood; but, on the other hand, it is certain that they may be moderately augmented in numbers under various conditions, including not only those enumerated by Virchow when (as I have already remarked) he proposed the term "leucocytosis," but also the process of suppuration or development of malignant new growths. Thus Dr Gowers mentions that the blood of a patient recovering from perityphlitis showed as many as 150 leucocytes in a not overcrowded field a few days before a large abscess appeared in the back. In 1875 a patient died in Guy's Hospital of jaundice from a cancerous tumour in the head of the pancreas, attended with suppuration in the liver. The blood was repeatedly examined during the last fourteen days of his life, and was always found to contain a decided excess of leucocytes,—as many as 130 to 150 in a field,—which were in active movement.

The *pathology* of splenic leuchæmia is still very imperfectly understood. Many observers, basing their opinions on the admitted doctrine that some at least of the white corpuscles of the blood are normally derived from the spleen, have imagined that they could account for the phenomena of the disease by supposing that the organ being enlarged, throws off an excessive number of white corpuscles. But this view ill accords with the fact that the total quantity of corpuscles in the blood is actually less than it should be, and that the red discs are greatly diminished in numbers. And it seems impossible to escape the conclusion that there is a change in the leucocytes themselves, which prevents them from undergoing conversion into red discs, as many of them naturally should. It is obvious that this in itself may be the starting-point in the whole morbid process. But if so, one must suppose that the formation of red discs,—at least as much of it as undergoes interruption in splenic leuchæmia,—has its normal seat in the spleen, since the disease could otherwise hardly be related, as we have seen that it is related, towards a previous attack of ague. The lesions in the liver, the bones, and other organs and tissues of the body may, notwithstanding Dr Cavafy's observations, be plausibly ascribed to the infiltration of superfluous leucocytes into their interstices; for there is certainly as yet no proof that these bodies constantly, and from the very first, are so devoid of vitality as to be unable to penetrate the wall of a capillary. Even the overgrowth of the spleen itself may be accounted for on the hypothesis that a large number of them are retained in its texture; and the occurrence of enlargement without leuchæmia may be attributed to a retention in the organ of all the leucocytes that fail to undergo the normal transformation into red discs; while, conversely, the fact that the blood is sometimes loaded with them, the spleen remaining of normal size, may be supposed to depend on their all escaping.

The *course* of splenic leuchæmia is generally one of slow progress towards a fatal termination. Its duration is commonly from one to three years; when it occurs in a child it is much shorter. The prognosis in an individual case must be based upon a careful study of the various symptoms; the extent to which the spleen is enlarged is of much less significance than the degree of anæmia and of breathlessness. As regards the state of the blood it probably matters much less whether the number of leucocytes is greatly increased than whether that of red discs is greatly diminished. Death often takes place unexpectedly by some complication, such as hæmorrhage, pleurisy with effusion, chronic peritonitis, phthisis or diarrhœa. Sometimes it is almost sudden, as in a case which occurred at Guy's in 1876, and in which œdema of the larynx was found at the autopsy.

Treatment.—There is reason to believe that at an early stage the disease may be arrested by treatment. Mosler relates the case of a boy, aged ten, whose spleen was considerably enlarged, and whose blood contained leucocytes in the proportion of one to twenty red discs; he took a drachm and a half of sulphate of quinine in the course of four days, and then ten grains and afterwards six grains daily; he completely recovered. Dr Goodhart, in 1876, stated to the Clinical Society that in the previous two years he had seen six cases, all in children under two years old, with moderate increase of the spleen, and with about ten times the usual proportion of leucocytes in the blood, and that they all got better under treatment, the medicine used being either phosphorus or the iodide of iron or cod-liver oil. Other measures which have been recommended are a cold douche directed upon the left hypochondrium, and the application of a galvanic or even of a faradic current to that part of the body, the positive pole being placed over the tenth rib, the negative over the enlarged spleen; it is said that by either plan of treatment the organ may often be greatly reduced in size.

On the other hand, when a case is already far advanced, little or nothing can be done to check its progress. Quinine is given without any appreciable result, and iron is equally useless. The only medicine which has sometimes appeared to benefit the patient is phosphorus. The transfusion of blood into his veins is hardly likely to be of any avail. Excision of the spleen should, I think, be rejected on account of the danger of rapid death by hæmorrhage. Arsenic has been tried and found useful.

Hodgkin's Disease.—In discussing the subject of tumours, I have, under the head of lymphoma (vol. i, p. 98) remarked that at the bedside, and even in the post-mortem room, one is obliged to class together under a single name certain cases in which the lymphatic glands, the spleen, and sometimes almost all the other organs and tissues, become the seats of growths which in different instances vary widely in their histological characters; they may be pure lymphomata, or they may be sarcomata, or they may occupy an intermediate position in the scale, so as fairly to deserve the title of lympho-sarcomata. As a rule, the cases in question present clinically a common group of symptoms; namely, a more or less general and sometimes extreme enlargement of the lymphatic glands, a moderate increase in the size of the spleen, with the presence of scattered nodules in it, marked anæmia, and a tendency to subcutaneous œdema of the face as well as of other parts, so that the patient's appearance is very like that of a person suffering from acute Bright's disease. They want, therefore, a designation which shall leave an open question their precise pathological anatomy, undeterminable as it is during life, and unessential as it seems to be even after death. Such a designation, and one which appears likely to meet with general acceptance, is that of Hodgkin's disease, proposed in 1865 by Dr Wilks, to whom we are mainly indebted for the recognition of the malady in this country, in place of the more vague title of "anæmia lymphatica," which he had given to it in an earlier paper in the 'Guy's Hosp. Reports' for 1862. He found, in fact, that his own observations, which were original, had been anticipated by Dr Hodgkin, a former lecturer on Pathology at his own school. In a communication made by that accomplished physician to the Royal Medical and Chirurgical Society in 1832, there are recorded several instances in which the spleen and lymphatic glands were jointly affected; and two at least of them, but more probably four, are examples of the disease now to be described. In Germany Virchow and Wunderlich may be especially mentioned as having written on it; and in France Trousseau, who gave to it the name of "Adénie."

With regard to the *causes* of Hodgkin's disease, but little is known. A very large proportion of those who are affected by it are children or young adults. Thus of seventeen cases that have ended fatally in Guy's Hospital since 1856, all but one have been in persons between eight years and thirty years old at the time of death; of the exceptions one was in a woman, aged forty-five, the other was in a man, aged fifty-six. A series of cases collected by Dr Gowers for his article in Reynolds' 'System' also shows a marked immunity in those who are between thirty and fifty; but among them the number of patients between fifty and sixty was remarkably large. At Guy's there have been fourteen males to three females. Dr Gowers gives the proportion as 75 to 25. Trousseau thought that there was often a starting-point for the glandular affection in some acute or chronic irritation at the angle of the eye, or in the external ear; and I find that we have had six cases in which there was some evidence of such a local origin. In one boy the swelling began in the neck, and was said to have followed a blow from a cricket ball; a girl had had suppuration of the ear, and indeed died of secondary meningitis; in a young man the development of the disease was preceded by abscess of the cervical glands; in two boys it followed measles, that exanthem having led in one instance to an abscess under the left side of the lower jaw, in the other to a swelling in a similar position which for a time subsided, but afterwards seemed to return. In a young man, in whom it began with enlargement of the glands in the left groin, there had been a chancre six months previously. Dr Gowers mentions the irritation of a decayed tooth as another possible cause for the disease.

As is implied in the preceding paragraph, there have been many cases in which an overgrowth of lymphatic glands in some particular region on one side of the body has been present for two or three years before there was any sign of extension of the disease to other parts. Dr Gowers alludes to the case of a boy whose axillary glands were excised by Mr C. Heath six years after they first became enlarged, and in whom, four years later, the cervical glands in the same side had alone become affected. And sometimes the morbid change has seemed to spread by continuity, as from the cervical glands to the thoracic, or from the inguinal to the lumbar. But in other instances it has apparently developed itself symmetrically on opposite sides of the body, as in both sets of axillary glands; or it may even have sprung up simultaneously in the most distant parts. One very striking character is that the affected glands, even when they have reached the size of pigeons' eggs, often remain isolated from, and freely moveable upon, one another, and unattached to the skin. They are commonly neither painful nor tender. But after a time they are apt to become fused together, either by a process of periadenitis, or more often by an extension of tumour-growth through their capsules, from one gland to another. They sometimes reach an enormous size; there may be several packets in different regions, each of the size of a child's head. Dr Gowers mentions a case in which they were found after death to weigh as much as 10 lbs. They are generally firm and elastic to the touch, but they may be very soft. A case of Bonfils' is cited in which an abundant quantity of lymph accumulated in one of the glands, and when a puncture was made, continued to escape from it.

But in other instances a general failure of the health precedes the development of any external glandular swellings. One cannot help suspecting that the affection has then really begun in some of the deeper glands, which are beyond the reach of manipulation; and this idea seems to be confirmed by a case, recorded by Dr Wilks, of a man who died in Guy's Hospital in 1856 in an extremely debilitated and anæmic state, with an enlarged spleen, and in whom the autopsy showed that the mediastinal and the lumbar glands were very much enlarged, although the superficial glands were all healthy. In such cases a doubtful diagnosis might perhaps sometimes be cleared up by the recognition of pressure-signs of one kind or another; for example, spasmodic cough and dyspnœa, distension of the veins, œdema of the arms or of the legs, or pain in the course of the lumbar or sacral nerves. Dr Gowers some years ago made an autopsy in a case of Sir William Jenner's, in which, there being general glandular enlargement, a mass of growth extended from the abdominal glands, and involved the solar plexus and nerves going to the adrenal bodies, so as to produce a discolouration of the skin having the distribution of Addison's disease, notwithstanding that the adrenals themselves were healthy.

Even glands lying outside the great visceral cavities sometimes interfere seriously with adjacent structures when much enlarged. In the neck they may compress the trachea or the œsophagus, and cause suffocation or a complete inability to swallow food; or, it is said, they may give rise to severe cerebral symptoms by pressing on the great cervical vessels, or to irregularity of the heart's action by involving the pneumogastric nerves. Enlarged glands in the neck may hamper very greatly the movements of the lower jaw in mastication. In the armpit they may compress the axillary vessels and nerves, so as to cause much pain in, and swelling of, the arm.

Pallor almost always becomes, sooner or later, a very conspicuous symptom in Hodgkin's disease. The cheeks and the lips appear bloodless and waxy looking; and the more so, as there is generally a very considerable degree of subcutaneous œdema, the eyelids being swollen, and the aspect generally being very like that of a patient with acute Bright's disease. It must be added, however, that albuminuria has sometimes actually been present as a complication, and that the kidneys have then been found after death to present diffused inflammatory changes in their cortical substance.

The state of *the blood* is commonly that of simple anæmia. When drawn, it is strikingly pale, and it has been compared to diluted claret; it coagulates slowly and imperfectly. Dr Gowers has found the red discs reduced to 60 per cent. of the normal proportion; in a case that occurred at Guy's Hospital in 1877 they were estimated at 76 per cent. The number of leucocytes is, in the large majority of cases, perfectly normal; but sometimes there is a slight increase of them, and in some rare instances the excess is enormous, rivalling that which is found in all but the most extreme cases of splenic leuchæmia. At least it is difficult to know what other interpretation can be set upon the various observations cited by Dr Gowers in a footnote to his article; he adduces no fewer than eight observations of extreme leuchæmia with enlarged glands and a spleen of normal size; two in which there was overgrowth of splenic pulp, but in which the organ contained no conspicuous growths; and two in which even this last character was present, the proportion of leucocytes to red discs being, in all the last four cases, that of one to four red discs. I think it is probable, however, that the occurrence of leuchæmia in all these cases should be regarded rather as an accidental circumstance than as an essential feature of the disease; just as a similar state of the blood is sometimes observed as the result of the growth of round-cell sarcomata in various parts of the body. And, as I have already remarked at p. 328, it undoubtedly sometimes happens that splenic leuchæmia and Hodgkin's disease appear to be associated together in the same patient. Thus in the 'Pathological Trans.' for 1873, a case of Dr Frederick Taylor's is recorded in which I made an autopsy and found that the spleen weighed fifty-one ounces, had quite the appearance usually seen in ordinary cases of leuchæmia, but that there were mediastinal and subpleural lymphomatous growths of very remarkable size.

The usual symptoms of severe anæmia accompany Hodgkin's disease. The respirations are accelerated, being often from 24 to 36 in the minute; there is dyspnœa, which on exertion may become extremely distressing. Epistaxis and purpura sometimes appear, but much less frequently than in splenic leuchæmia. The temperature of the body often rises to 100° or higher; in one instance I have known it reach 103·2°. According to Dr Gowers, the pyrexia is sometimes continuous, with slight diurnal fluctuations; sometimes it lasts only for a few days at a time, the febrile periods being separated from one another by intervals during which the temperature is normal; sometimes it is especially characterised by morning remissions, the daily range amounting to 3° or even more. In 1860 a man died in Guy's Hospital nine days after his admission, in whose case no diagnosis was made during life; he lay with his eyes closed, and he was scarcely sensible; his skin was hot, and he had occasional rigors; his spleen could just be felt; he had diarrhœa; there was, indeed, a mass of enlarged glands in the left side of the neck, but I suppose this was thought accidental and without relation to his acute illness, for the opinions expressed were that the case was either one of ague or of enteric fever; the autopsy showed that it was one of Hodgkin's disease. A persistent high temperature appears to be a sign which, more than any other, indicates that the patient's life will rapidly be brought to an end.

The *course* taken by cases of Hodgkin's disease is, indeed, very variable. Sometimes the patient is known to be ill during only a very short period before the occurrence of a fatal termination; and the lymphatic glands may then undergo a very rapid enlargement. Thus in 1867 a man, aged thirty, came under my care, whose case I have already recorded in the 'Guy's Hospital Reports' for 1881. Except that a month previously he had a slight cough and hæmoptysis (which probably were due to tubercular disease of the lungs, since this was found to be present at the autopsy) he was supposed to be well, and remained at his occupation as a hawker of fish until three weeks before his death. He was then suddenly seized with a dull heavy pain at the chest, and six days later, on March 16th, he became covered with purpuric spots. On the 21st hæmaturia set in. He also expectorated a quantity of blood, which seemed to come from the mouth. He was admitted on March 23rd. The spleen was then much increased in size, its edge being felt about half an inch below the ribs; but no enlargement of lymphatic glands was discovered. During the next few days his temperature ranged from 99·4° to 99·9°. The diagnosis was "purpura hæmorrhagica." On the 28th he had epistaxis. On the morning of the 30th at about 7 a.m. he noticed, for the first time, that the glands of his neck were enlarged and very tender. He pointed this condition out to the clinical clerk, and it was then found that all the other glands of the body were likewise swollen, although not so tender. Notwithstanding that he was perspiring freely, his temperature was 103°. Extreme dyspnœa set in two days later, on April 1st; and he died, suffocated by œdema of the larynx, at noon on that day. On post-mortem examination the cervical and the axillary glands were seen to be enlarged, so that some of them measured an inch in their long diameter; they were soft, of a pinkish cream colour, and spotted with ecchymoses. The tonsils presented a similar appearance, and were half an inch thick. The thymus formed a large pear-shaped mass. The spleen weighed twenty ounces; it was pale and soft. The kidneys were very pale and spotted all over with patches which looked as though they were suppurating. Dr Moxon found distinct evidence of leuchæmia: leucocytes were visible in large numbers in the liver between the hepatic cells, and as many as twenty-five were counted in a single short capillary vessel of the substance of the heart. On the other hand, I had examined the blood a day or two before the patient's death, and had failed to discover any excess of white corpuscles. Thus the case rather tends to confirm the opinion expressed above that leuchæmia is no essential failure of the disease even when it is present.

Two cases somewhat similar were recorded by Dr Paterson in the 'Edinburgh Medical Journal' for 1870. The first was that of a young woman, aged twenty, who having previously been plump and well-coloured, became towards the end of a first pregnancy very sallow, with hollow eyes, although she still said she felt well and in good spirits. Her confinement was followed by troublesome hæmorrhage, which, however, readily yielded to ergot. About the sixth day afterwards a marked change for the worse took place. The pulse became rapid, there was considerable heat of skin, the liver and the spleen were found to be enlarged, and the glands in the neck were also slightly increased in size. The blood was now examined and it proved to be highly leuchæmic. Death occurred only five days later from suffocation, the cervical glands having in the meantime reached a great size, and the dysphagia having been so extreme that she could not swallow even a teaspoonful of water. Dr Paterson's second case occurred in a policeman's wife, who had become languid, pale, and sallow during the latter part of her first pregnancy, and who had considerable hæmorrhage after delivery. Soon afterwards a slight increase in size of the cervical glands was detected, and it was also found that there was leuchæmia, and that the liver and the spleen were enlarged. The glands of the throat and neck and upper part of the chest underwent further increase, fever and restlessness set in, and she died of asphyxia when her baby was only fourteen days old. There was no autopsy in either case.

As a rule, however, the progress of the disease towards a fatal termination is slow. Dr Gowers gives a table showing the duration of fifty fatal cases, the length of which could be fixed with some degree of accuracy; thirty-three of them ended within two years. The most common mode of death is gradual exhaustion. But, as we have seen, suffocation sometimes occurs from pressure of the enlarged glands upon the trachea; and sometimes starvation, from interference with the œsophagus. Epistaxis has occasionally been directly fatal. Coma, delirium, and convulsions, without discoverable pathological cause, have been observed in some cases by Dr Southey. Pneumonia, œdema of the lungs, and pleurisy are not infrequent complications, and may be the immediate causes of death. Diphtheria of the fauces seems to have occurred more often than can be accounted for on the supposition of a mere coincidence.

Anatomy.—The morbid anatomy of Hodgkin's disease varies in different cases. The affected glands usually appear whitish yellow, waxy, smooth, and firm, both on surface and on section, but sometimes they are opaque, white, soft, medullary, and perhaps spotted with hæmorrhages; and in one case that I examined they were of a uniform deep reddish-grey tint. They have remarkably little tendency to caseate.

The *spleen* is only moderately enlarged, its weight in the cases that have occurred at Guy's Hospital having varied from eight to twenty-eight ounces. On section it is found to have scattered through its substance a number of firm whitish-yellow masses, of round or irregular shape, from the size of peas to that of hazel-nuts. Dr Wilks has been accustomed to compare them to masses of suet in a pudding, or to the almonds in "hardbake." Sometimes the nodules present one or more concentric rings. The only instance that I know of in which such an affection of the spleen was unconnected with a diffused morbid change in the lymphatic glands was in the case of a man, aged sixty-seven, who died of cerebral hæmorrhage, and in whom it was accidentally found at the autopsy, the glands being normal both in size and in appearance.* But sometimes the spleen in Hodgkin's disease presents only an indefinite mottling, or its tissue may be uniformly red and homogeneous.

In other organs the growths are very variable in character. The *liver*, which may be greatly increased in size, sometimes contains distinct nodules, but more often it merely shows tracts of lymphoid tissue running along the portal canals, or minute nodules scattered between the lobules in such a way as to be distinctly recognised only with the microscope. Thus, the state of the liver is, I believe, often undistinguishable from that which occurs in splenic leuchæmia.† The *kidneys* present similar combinations of diffuse interstitial growth with more or less sharply defined and rounded tumours. The solitary *follicles* of the intestine and also Peyer's patches are sometimes greatly swollen and medullary-looking; and it is worthy of notice that the *tonsils* * and the follicles at the root of the tongue may be affected in the same way, since they can of course be seen during life to be enlarged. Another organ which is accessible to clinical investigation is the *testicle*. In Dr Taylor's case, already referred to, each epididymis was enlarged so as to be two or three times as big as the testicle itself; and a similar condition is mentioned as having been present in one of Hodgkin's original cases. Within the thorax there are sometimes enormous masses of growth. Dr Taylor's patient (in whom, as I have already observed, there was leuchæmia, with a very large spleen) had in the anterior mediastinum a flattened tumour, one inch thick, with the left innominate vein running through its centre. In that instance there were also in each parietal pleura large flat nodulated bands of lymphoid growth of red colour, running parallel with the ribs. In other cases the pericardium and the base of the heart have been invaded, or the disease has spread into the lungs from their roots. Not infrequently the *thymus* has been greatly enlarged and infiltrated with a soft white growth. The medullary tissue of the *bones* does not always escape. In a case which I recently examined I found in one tibia a soft rounded mass, as large as a nut, which, however, had not a lymphoid structure, but was made up of a glistening fibrillated matrix with nuclei embedded in it. Writers also describe a diffused change in the medulla which is said to have been converted into a reddish-grey semi-diffluent substance.

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* [Another is recorded in the 'Path. Trans.' for 1870, p. 390, in a girl of seventeen. There was also diphtheritic angina and colitis.—Ed.]

† [In a case under my care in 1874, the liver weighed 88 and the spleen 83 oz., without leuchæmia or enlargement of lymph-glands. 'Path. Trans.,' xxvi, p. 199.—Ed.]

Dr Wilks long ago suggested—and Virchow is disposed to admit it—that there is some relation between Hodgkin's disease and the lardaceous change. In two cases that have recently occurred at Guy's Hospital the change in question has been found in the viscera as well as in the enlarged glands, but each patient had had one or more surgical operations performed which had led to the formation of pus, though not in very large quantity.

Diagnosis.—The recognition of Hodgkin's disease is generally easy at an advanced stage; but at the commencement, when the only symptom is a mass of glands in the neck, in an armpit, or in one of the groins, it is probably never possible to deny that the case may turn out one of "simple lymphoma." And when the glands, instead of being freely moveable one over another, are matted together, one must bear in mind that the affection may perhaps be tuberculous, or syphilitic, or secondary to some deep-seated malignant tumour. On the other hand, the fact that degenerative changes, and even suppuration, should have occurred extensively in the packet of glands first affected, is not always a proof that the subsequent progress of the case may not be that of Hodgkin's disease rather than of a diffused tuberculosis. As regards syphilis it is perhaps worth while to quote the case of a man who was admitted into Guy's Hospital in 1867, having in the left side of the neck, from the occiput to the shoulder, a tumour which was believed by Dr Moxon to consist of a gummatous enlargement of the lymphatic glands; after death, however, it appeared to have its seat rather in the other tissues of the neck, the glands being embedded in it, but being themselves unaltered. As an instance of a secondary sarcoma of glands, the nature of which was unrecognised during life, I may mention the case of a girl, aged ten, whose body I examined in 1880, she having died immediately after an operation for the excision of a mass of glands in the left axilla and above the left clavicle. It turned out that there was a primary tumour in the left broad ligament, and that the lumbar glands were also sarcomatous, as well as one of the mediastinal glands, from which growth was extending into the right auricle of the heart.

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* [See a case by Dr Moxon, 'Path. Tr.,' xx, p. 369, and Dr Legg, 'St Barth. Hosp. Rep.,' xi. The "lenticular" lymph-follicles of the stomach are also enlarged in some cases (Virchow, 'Krankh. Geschw.,' p. 509), and the agminated lymph-follicles of Peyer (Heschl, quoted in 'Virchow's Gesammelte Abh.,' p. 199, and Béhier, in a monograph on 'Leucémie intestinale,' 1868).—Ed.]

Treatment.—In spite of such cases as these, I am inclined to think that it is well to have such glandular tumours excised as early as possible. Verneuil has recorded one striking case in which an immense mass was removed with perfect success, and the patient was still in good health seven years afterwards.

Of internal medicines, *arsenic* and *phosphorus* appear to be the most likely to do good. The reputation of arsenic rests mainly upon a case of Billroth's, in which the disease had existed for ten months, the patient being a woman of forty; the cervical, the axillary, and the inguinal glands were greatly enlarged; within a fortnight after the commencement of the treatment they were already reduced in size, and after two months she was discharged with only a single gland of the size of a nut on each side of the neck. No similar success has been obtained by other observers.* Phosphorus was first given by Verneuil; Dr Gowers in one case found that its administration was followed by a remarkable diminution in the size of the glands, and by a reduction of the proportion, which had been excessive, of leucocytes in the blood, but the patient died of disease of the kidneys, to which, Dr Gowers thought, the medicine possibly contributed. It must be borne in mind that the glands have sometimes become very much smaller shortly before death, even when no active treatment had been adopted.

PROGRESSIVE DIFFUSED TUBERCULOUS DISEASE OF THE LYMPHATIC GLANDS.—In discussing the subject of tubercle, I have already briefly alluded to tuberculous affections of the lymph-glands (vol. i, p. 79), and I have described the symptoms which are observed when the bronchial and the mesenteric glands respectively are attacked by disease of this nature. The corresponding lesions of the cervical, axillary, and other external glands come rather under the province of the surgeon.

But instances are not very infrequent in which nearly all the glands in the body are simultaneously, or in rapid succession, affected with tubercle, and in which a severe and fatal illness results. A case in point, which has sometimes been wrongly cited as an example of Hodgkin's disease, was recorded more than a century ago by Morgagni (Epist. lxviii).

I find no fewer than ten such cases in the reports of post-mortem examinations at Guy's Hospital during the last fifteen years. Two or three of them have already been published. Thus Dr Goodhart related in the 'Guy's Hospital Reports' for 1873, the case of a man who was admitted under Mr Cooper Forster for disease of the right knee-joint, and who also had a large mass of glands in the right posterior triangle of the neck. He died after amputation of the thigh. At the autopsy the glands in Scarpa's triangle on the affected side were found to be enlarged, yellow, and of putty-like consistence. The inguinal, the iliac, and the lumbar glands on the right side were all very large, firm, and yellow, those on the left side being healthy. Some of the glands in the portal fissure were as big as chestnuts. The bronchial glands on both sides were much enlarged, some being more than an inch long; they were cheesy, and disease seemed to be spreading from them into the lung itself on the left side, in the form of rounded yellow masses. The glands in the right axilla, and those on both sides of the neck, were in a similar condition. There was also miliary tuberculosis of the lungs. Another case was recorded by myself in vol. xxv of the 'Path. Trans.,' at p. 235. A woman, aged thirty-five, was admitted with a large suppurating glandular swelling in the left groin, and with another mass of swollen glands in the left side of the neck. She said she had been gradually wasting since her marriage two years before. She died at the end of a week. At the autopsy it was found that along the whole length of the spine there was a continuous mass of enlarged and suppurating glands. The aorta and the vena cava ran through a dense agglomeration of glands, some more than an inch long, many of which contained points of pus, while some were even sloughing in the centre. The portal glands and the bronchial glands were similarly affected. The right inguinal and the right iliac glands, however, were in an earlier stage and looked greyish white and granular on section. The axillary glands were little, if at all, involved in this disease. The spleen weighed twenty-four and a half ounces; it contained numerous yellow masses, some as large as walnuts. The lungs showed a few masses of the size of peas, but no ordinary tubercles; nor were any tubercles present in the intestines nor in the liver. A third case was that of a man, aged twenty-seven, admitted into a surgical ward on account of his having a swelling in one axilla of the size of a hen's egg, attended with pricking pain; it was growing rapidly, but was moveable, and the skin over it, although red, was not adherent. It was excised and was found to consist of enlarged caseating and suppurating lymphatic glands. He died subsequently of pyæmia. At the autopsy I found that a similar state of the glands extended under the pectoral muscle and that all the mediastinal glands were affected; in the spleen there was a single tubercle softening into a little cavity. Of the remaining cases five were in men, aged respectively eighteen, twenty-four, thirty-five, thirty-seven, and fifty-four years; two in women, one aged thirty, the other forty-seven ('Path. Trans.,' xxvi, 202). In every instance, with one exception, it is noted that the spleen contained tubercles.

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* [Injection of Liquor arsenicalis into the enlarged glands by a subcutaneous syringe has also been practised at Vienna. I once tried it on a patient of my own, but it produced pain and inflammation without apparent benefit.—ED.]

The duration of this remarkable form of adult tuberculosis was generally from six months to a year. It was attended with fever, and with rapid wasting.

It must be of considerable practical importance to the surgeon to recognise the occurrence of cases of this kind, since he is very apt to be led to excise some prominent mass of glands from the neck or the axilla, a proceeding which is not likely to be attended with any satisfactory result. To the physician their chief importance lies in the diagnosis from Hodgkin's disease, with which they are generally confounded. It is interesting to note the great frequency with which tuberculosis of the spleen is met with in association with a similar affection of the lymphatic glands. This fact was long ago pointed out by Bright in vol. iii of the 'Guy's Hospital Reports.'

AFFECTIONS OF THE URINARY ORGANS

FUNCTIONAL DISORDERS

Polyuria and oliguria; high and low specific gravity—Diabetes insipidus—"Renal inadequacy"—Reaction: alkaline urine, and phosphatic deposits—Urobilin and Indican—Uric acid and urates—Oxalates—Cystine—Calcic sulphate—Hæmoglobinuria—Ammoniacal urine—Bacteruria.

In dealing with the affections of the urinary organs, I shall endeavour to carry out the same plan which I have adopted in other sections of this work, that of first taking those affections which are comparatively simple in their phenomena, and afterwards passing to those which are more complicated. Thus in the present chapter: (1) I shall describe certain morbid conditions of the urine that are independent of the presence of any organic lesion of the kidneys, being mostly characterised by some alterations in its *quantity*, *density*, or *reaction*, by the formation of *precipitates* or deposits, or by the occurrence of putrefactive changes. (2) Secondly, I shall consider the various morbid processes that may affect the renal pelvis or the ureter, in consequence of the presence of calculi, suppurative inflammation of the kidney, and pyelitis. Generally speaking, these affections are characterised by the presence of *blood*, or of *pus* in the urine. (3) The next chapter will be devoted to diabetes, a disease in which the urine contains *sugar*. (4) After this I shall discuss those renal affections which constitute Bright's disease; of these it is a common feature that *albumen* escapes through the glomeruli into the urine. (5) The last chapter will treat of tubercle and new growths of the kidney, with parasites, &c.

There are many simple morbid conditions of the urine, such as the absence of chlorides in pneumonia (vol. i, p. 900), and the presence of bile-pigment in jaundice (vol. ii, p. 260), or of leucine and of tyrosin in acute yellow atrophy of the liver (p. 265), which require no account in this place, because they were fully described when those diseases were being discussed. What I have now to do is to give an account of the changes in the urine that do not belong to any other well-marked pathological process, but constitute, from a clinical point of view, so many independent affections. Among these there is theoretically to be found a line of distinction which it would be very desirable to draw broadly and clearly. In some the urine contains an abnormal material, or a normal material in undue quantity, as the result of a morbid process in the system generally or in organs remote from the kidneys. In others the formation of a deposit is due merely to a relative excess or deficiency of the substances which give the urine an acid reaction, or to the occurrence of fermentative changes in it after it has been secreted. Unfortunately, however, one cannot carry out this division completely; for in many cases, as for example with regard to oxalate of lime and uric acid, there is the greatest difficulty in determining whether the urine does or does not really contain too much of the materials which are thrown down from it as deposits. But in dealing with my subject I shall, as far as possible, adopt such an order as will bring out, first those affections in which the urine is faulty from the time of its formation, and afterwards those in which its morbid condition is traceable more or less completely to changes occurring in it when it has already passed into the pelvis of the kidneys, or even into the bladder.

Changes in the quantity and in the density of the urine.—None of the other characters of the urine appear so fundamentally important as its quantity and its specific gravity or density. And therefore it may be convenient to begin by discussing certain affections of which the essential feature is that the renal secretion is altered from the normal in these respects. Between quantity and density there is an inverse proportion. In health, when the urine is very abundant, it is always pale and watery; when it is scanty it is dark, and of high specific gravity. And so among diseases, diabetes, in which sugar is drained off from the blood in large quantity, affords the only case in which urine secreted in excess is also of great density.

On the other hand, a diminished flow of urine is not likely to be accompanied with a low specific gravity of the secretion except when the substance of the kidneys has undergone extensive destruction as the result of advanced Bright's disease. In normal circumstances, the daily amount of urine is generally said to range in different individuals from about forty to fifty ounces; it is liable to great variation from day to day, and probably there are some perfectly healthy individuals who habitually void quantities which may be either considerably larger or considerably smaller. The specific gravity of the twenty-four hours' urine generally varies between 1015 and 1025.

Physiology teaches that the quantity and the density of the urine depend mainly upon the activity of the blood-current in the renal glomeruli. According to Ludwig the determining factor is the pressure of the blood within the vessels of the tufts; according to Heidenhain it is rather the rapidity of its passage through them. The chief point in favour of the latter theory is the fact that when in experiments on animals the outflow of blood through the renal veins is checked, the urine becomes scanty and of high specific gravity, but Cohnheim shows that this fact is perhaps not so completely inconsistent with Ludwig's view as might at first sight appear. Clinically, the distinction appears to have but little significance. The only local cause of obstruction of the renal veins, as a morbid condition, is thrombosis of these veins or of the inferior cava above their mouths. Now, thrombosis of the renal veins is not very infrequent as a complication of lardaceous and other forms of Bright's disease, and Dr Moxon has recorded in the 'Guy's Hospital Reports' for 1869 two cases in which it was associated with injuries to the lumbar spine. But in the former class of cases the existence of lesions in the renal cortex makes it impossible to determine the effect of the thrombosis upon the characters of the renal secretion, and moreover as the obstruction is probably developed very slowly and gradually, collateral channels have time to enlarge and can carry on the circulation. And in both of Dr Moxon's cases the arteries were plugged as well as the veins, so that the urinary secretion was of course entirely suppressed. Bartels has related in Ziemssen's 'Handbuch,' a case in which the inferior vena cava was closed by thrombus from the point where it passes through the groove in the back of the liver downwards; in that instance, however, the urine, which contained blood and albumen, was secreted in fair quantity, and was of sp. gr. 1011 to 1013.

On the other hand, when the systemic venous circulation generally is obstructed, as in cases of heart disease and of pulmonary emphysema, the urine is almost constantly found to be scanty and of high density. But this accords equally well with Ludwig's and with Heidenhain's theory, since the pressure in the arteries is in such circumstances lowered as the result of enfeeblement of the left ventricle. When under the influence of digitalis the arterial pressure can be brought up to a sufficient point, it is surprising how greatly the secretion of the kidneys becomes increased. There is not, so far as I know, any primary morbid state of which it is an essential feature that the urine should be deficient in quantity and of excessive density. Dr William Roberts, however, relates the case of a man, aged fifty, who for several days passed only about thirty ounces of urine daily, of specific gravity 1028 or 1029, containing an amount of urea which was at least a quarter above the average for his body weight, this being only 8 st. 6 lbs. At one time there was a small quantity of sugar in the urine. Prout thought that he recognised a disease, which has since been termed *azoturia,* the fundamental symptom of which was an increase in the excretion of urea. In cases which have been placed under this category the flow of urine likewise has been generally rather excessive. But it seems very doubtful whether they have been rightly interpreted.

A state of urine exactly opposite to that which has been mainly discussed in the last paragraph,—the quantity being augmented, and the density reduced, is of frequent occurrence. We shall find it to be a very important and often an early symptom of certain forms of Bright's disease, being then dependent upon the abnormally high arterial tension that characterises that disease. But it is also seen as an independent affection.

Diabetes Insipidus.—In this disease the patient passes enormous quantities of urine, exceeding even those that are voided in saccharine diabetes itself, for which it is pretty sure to be mistaken until chemical analysis shows that no sugar is present. The specific gravity constitutes another marked distinction between the two affections, for in diabetes insipidus it is often scarcely above that of water, and seldom reaches higher than from 1003 to 1007. The daily secretion of urine may range from fifteen to thirty and even forty pints. It is clear and almost, if not quite, colourless; it has a faintly acid reaction, but undergoes the ammoniacal fermentation rather early. It of course contains but a very small proportion of solid matters, but nevertheless the total daily amount of urea excreted appears to be rather excessive than diminished.

One abnormal material, *inosite,* has been sometimes detected in it; the chief test for this substance, when dissolved, is that of Scherer, which consists in evaporating cautiously to dryness, moistening the residue with ammonia and with solution of chloride of calcium, and then evaporating again, whereupon a rose-red colour makes its appearance. There is no reason to suppose that the presence of inosite is an essential feature of diabetes insipidus. In fact, it is often absent in this disease; and on the other hand it has been sometimes detected in saccharine diabetes and also in Bright's disease. As it is always to be found in small quantity in the muscles and in the lungs, liver, spleen, and other organs, the supposition has been hazarded that its excretion in the urine may be the result of the excessive transudation of water through the tissues of the body. Strauss is said to have discovered it in the urine of three healthy persons, each of whom, for the purpose of experiment, had drunk a very large quantity of water. But on this view it is difficult to see why, in diabetes insipidus, inosite should ever be wanting. Lastly, notwithstanding that the absence of sugar constitutes, in general, the main distinction from saccharine diabetes, it is the fact that minute quantities of sugar have been present in the urine in some exceptional cases which in other respects have appeared to be examples of diabetes insipidus.

A tormenting thirst is one of the main symptoms. At one time it was imagined that this might perhaps really constitute the essential feature of the disease, which should therefore properly be regarded as a "polydipsia," rather than a "polyuria." But experience has shown that the patients always pass more urine than healthy persons who drink the same quantities of fluid, and moreover when in a case of diabetes insipidus the amount of drink is restricted, the urine does not fall in the same proportion, and the tissues become dry. So severe is the thirst that patients who have not been allowed to satisfy it have been known to drink up their own urine, as in a case related by Trousseau.

In some instances the general health remains wonderfully good. Dr Wm. Roberts cites the case of a farm labourer, aged fifty-one, who had been affected for twenty-four years, drinking from thirty-two to thirty-six pints of water daily, and voiding urine in proportion, and who yet remained able to do all kinds of hard work, such as thrashing and mowing. And another case is recorded of a woman, who bore eleven children while suffering from the disease. It is especially noted that the farm labourer's skin was moist, and that he perspired freely when at work. But, as a rule, the skin is obviously dry and harsh. Dr Roberts himself had under his care a boy, who, except that his skin and his tongue were dry, appeared pretty well, looking rosy and plump. Most patients, however, complain a good deal. One thing which troubles them is that their rest at night is disturbed by the frequent desire to micturate. Other symptoms are, according to Dr Roberts, a painful dryness and heat of the mouth and fauces, pains in the loins and in the epigastrium, an indifferent or sometimes a voracious appetite, enfeeblement of bodily strength and of mental vigour, irritability of temper, abolition of the sexual functions. Senator, in Ziemssen's 'Handbuch,' states that the temperature of the body is slightly lowered, the large quantities of fluid that are swallowed having a cooling effect. Enforced abstinence from drink aggravates most of the symptoms; the body then feels unnaturally hot, the skin is suffused, there is an intolerable sense of sinking, or even intense pain at the pit of the stomach, the intellect becomes impaired. Sir Thomas Watson relates the case of a boy, aged eleven, who was limited during twenty-four hours to drinking a pint and a half of fluid, and who nevertheless passed ten and a half pints of urine. That he absorbed water from the air seemed to be clear from the result of weighing him at short intervals. Thus one day, after having just emptied his bladder, he weighed 3 st. 8 lb. 0 oz. 3 dr. Three hours later, having taken nothing in the interval, he weighed 3 st. 9 lb. 0 oz. 2 dr. Then he voided 16 oz. of urine, and after this his weight was again 3 st. 8 lb. 0 oz. 3 dr.

Diagnosis.—Diabetes insipidus is of infrequent occurrence as compared with most other diseases; in London hospital practice I should say that it is decidedly rare. And with regard to all published statistics there is a doubt whether they are not to a greater or less extent vitiated by the inclusion of cases in which some form of Bright's disease would have been found present if an autopsy had been made. Dr Roberts, indeed, avowedly places in his collection of seventy-seven cases three which ended fatally, although in each of them the kidneys were affected with a marked degree of atrophy in association with hydronephrosis, or (as I shall term it) "consecutive Bright's disease." Now, it is clearly important to distinguish from diabetes insipidus all cases in which the polyuria, however excessive, is a mere symptom of an organic lesion of the kidneys. But the question is whether, in any of the cases in question (two of which came under the observation of Dr Eade, of Norwich), the hydronephrosis could itself have been a secondary result of the frequent micturition, just as we shall find hypertrophy of the bladder to be a not infrequent consequence of saccharine diabetes. In one instance the patient, a man aged sixty-two, is said to have suffered for twenty years from excessive thirst and diuresis. For my own part, I think that the clinical diagnosis of diabetes insipidus should always be regarded as somewhat uncertain in persons advanced in years, on account of the insidious way in which renal cirrhosis comes on, often without producing any albuminuria. Probably, however, mistakes might be prevented by careful observation of the state of the arterial tension, which in diabetes insipidus appears to be lowered rather than excessive. Even in young subjects one must not overlook the possibility of the presence of hydronephrosis from calculous disease in childhood.

Ætiology.—Among Dr Roberts's cases there were seven in which diabetes insipidus was said to have begun in infancy (in two or three actually from the time of birth), fifteen in which it began between the ages of five and ten years, thirteen between ten and twenty years, sixteen between twenty and thirty years, fifteen between thirty and fifty, and four between fifty and seventy. Males preponderated over females in the proportion of two and a half to one.

In a few instances there was a well-marked history of the occurrence of the disease in several members of the same family; the most striking example of this seems to be one recorded by Lacombe, in which a mother, her three sons, her daughter, her brother and his children, were affected in turn. According to Trousseau, diabetes insipidus is not uncommonly seen in persons whose parents had suffered from diabetes mellitus, or from albuminuria. In some cases it has been attributed to trivial circumstances, such as exposure to cold or heat, drinking cold fluids when heated, intemperance, muscular efforts, mental emotions. But the most important of its exciting causes are blows or falls upon the head, and certain organic lesions of the brain. As regards the traumatic cases, Dr Roberts remarks that in some of them the polyuria has set in with its maximum intensity on the very day of the accident, but in others not until after the first loss of consciousness had passed off, or a few days later, or even in one case not until the time of subsidence of severe nervous symptoms at the expiration of six months. In some of the cases associated with cerebral lesions there have been scrofulous or other tumours, occupying various positions,* but in one instance projecting into the fourth ventricle from its floor. In two cases an appearance described as "fatty degeneration" of the nerve-cells in the neighbourhood of the fourth ventricle was noted. But it may perhaps be a question whether the supposed lesion really had as much significance as was imagined, for I believe that it is not very infrequently found in cases in which the brain may be presumed to be healthy.

Such observations possess special interest in consequence of the fact that an affection like diabetes insipidus can be produced experimentally in animals by injuries to certain parts of the nervous apparatus. Bernard first showed that this effect followed puncture of the floor of the fourth ventricle at a point a little above that at which glycosuria is generated. There is also experimental evidence as to the production of an excessive flow of urine by irritation of the cervical sympathetic and by lesions of the spinal cord. The probability seems to be that the immediate cause of the affection is a dilatation of the renal arteries, from defect of the controlling action of their vaso-motor nerves. So far as is yet known there are no special secretory nerve-fibres influencing the renal function. In those cases in which the urine contains a trace of sugar, it has been conjectured that there may be some morbid state of the two contiguous centres in the bulb, which are respectively associated with diabetes insipidus and diabetes mellitus. And it is worthy of mention that in a case observed by Külz, diabetes insipidus was accompanied by spontaneous persistent ptyalism (the patient spitting from twelve to eighteen ounces of saliva daily); for this is another symptom that has been produced in animals by puncture of the floor of the fourth ventricle.

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* Dr Alexander Hughes Bennett, 'Brit. Med. Journ.,' Feb. 24th, 1883.

Prognosis and treatment.—The course of diabetes insipidus varies widely in different cases. Dr Roberts cites one instance in which it set in with absolute suddenness; the patient, a woman, aged thirty-four, went to her work one morning at 6 a.m. in her usual health; two hours later she was seized with intense thirst and diuresis, which continued from that time. Some years ago I met with a remarkable case in a man living at Dulwich, who had several distinct but very short attacks of what appeared to be diabetes insipidus. I have unfortunately preserved no notes, but my impression is that each attack lasted a day or two, and I remember that he passed enormous quantities of urine, and became for the time exceedingly prostrate and exhausted. I think that this recurred at intervals of some weeks. In those cases which follow injuries to the head, the affection commonly subsides in the course of a few weeks or months, but cases are on record in which it had been of six or seven years' duration. And of the non-traumatic cases beginning from infancy, some are stated to have run on for fifty years or more. When there is a cerebral tumour this is of course almost sure to prove fatal in a comparatively short time. Otherwise, diabetes insipidus does not in itself appear to have much tendency to destroy life; for the patient generally succumbs to an intercurrent malady, such as phthisis or pneumonia. It is a remarkable fact that the occurrence of some inflammatory or febrile disease has in several cases led to the temporary, or even permanent, subsidence of diabetes insipidus. Thus one patient who had suffered from it for eighteen years recovered completely after an attack of acute rheumatism, and another after an attack of pleurisy, treated by a blister which suppurated for thirty-five days. Dr Roberts suggests that it might be worth while in future cases to try the effect of a large blister applied to the back of the neck, or to the epigastrium. Among medicines the most useful seems to be valerian, which was prescribed by Trousseau in enormous doses, two and a half drachms of the extract daily, or even more. To one patient he gave nearly an ounce, and in the course of four months recovery took place. Dr Roberts relieved a boy under his care with the valerianite of zinc, increasing the dose until it reached twenty grains a day. Ergot is another remedy that has been used with more or less success. Galvanism (the constant current) has been recommended by some German observers. One pole may be applied to the loin on one side near the spine, and the other to the corresponding hypochondrium for five minutes; and then they may be transferred to the opposite side of the body in the same manner; or, as Külz advises, the positive pole may be placed upon the nape of the neck, the negative pole, first to the loins for four minutes, and then to the epigastrium for the same period of time.

Renal Inadequacy.—Under this name Sir Andrew Clark has described ('Brit. Med. Jour.,' i, 1883) a class of cases of which the main feature is that the kidneys appear to be unable to excrete more than the normal daily quantity of urine (from forty to fifty ounces), which yet has a low specific gravity (1002 or 1003 to 1008), and is deficient in urea (not containing more than 2 per cent. of it), though the amount of uric acid may be natural. Even if patients whose kidneys have this peculiarity as regards their functions drink freely of water, they do not pass a larger quantity of urine; and a liberal diet with a good allowance of wine is obviously injurious to them. The urine in such cases is devoid of albumen, and there are no casts in it. And although Sir Andrew Clark admits it to be possible that the kidneys are on the way towards chronic Bright's disease, he says that when he has had an opportunity of making an autopsy the organs have appeared to be healthy. The patients are generally ailing, without being definitely ill. One point about them is that they are apt to take cold, and do not get rid of the cold easily, being also liable to be attacked with pneumonia, pleurisy, and pericarditis without apparent reason. They recover slowly from even slight injuries, and they bear surgical operations badly, a fact which appears to have been noticed independently by Sir James Paget. They complain of *malaise*, and weakness, and unfitness for work; they sleep badly, are subject to headache, and suffer from nervousness. Sir Andrew Clark does not say anything about the state of the arterial tension in these cases. Ultimately he describes patients so affected as developing a condition very like myxœdema, with puffy pink and white faces, a dry glossy skin, a slow articulation, and a somewhat staggering gait. The main points in the plan of treatment which he advises for them are a very sparing diet and careful management of the skin. He allows for breakfast bread and butter and an egg; for the midday dinner not more than half a pound of meat with vegetables, and afterwards some pudding; about six or seven o'clock, bread and butter again with an egg, or a very little fish, or the wing of a chicken.

Reaction of the Urine.—Urine has normally an acid reaction, which is commonly said to be due to the presence of the acid phosphates of soda and potass rather than of free acid. But that this view is, after all, arbitrary, appears to be shown by an experiment devised by Malz and Donatti, as cited by Salkowski. It consists in dissolving in a solution of the neutral phosphate of soda (Na_2 HPO_4) the amount of hippuric acid necessary to combine with one equivalent of the sodium. If now the resulting fluid, which is strongly acid, be shaken up with ether, hippuric acid is extracted, and the fluid gradually regains the alkaline reaction that belongs (in spite of its name) to the neutral phosphate. But if, on the other hand, the fluid be dried, and the residue extracted with ether, no hippuric acid comes away. These apparently contradictory results clearly indicate that the relations of bases to acids in such fluids are unstable, and vary with circumstances, so that no positive statement can be made about them. The degree of acidity in the urine (which must be estimated from the secretion collected through the whole twenty-four hours) is commonly expressed in terms either of the dried carbonate of soda required to neutralise it, or of an equivalent weight of oxalic acid. Dr Roberts found that in a healthy man it amounted on an average to about fourteen grains of the carbonate; but there are wide variations, the range during a period of nineteen days being from six to more than twenty-three grains. Writers who give it in terms of oxalic acid say that it corresponds to about thirty grains of that acid or more.

At different periods of the day, however, the reaction of the urine is by no means uniform. After each of the principal meals it becomes for a time decidedly less acid, and may even be alkaline. This fact was originally pointed out by the late Dr Bence Jones, and has been fully confirmed by Dr Roberts. According to the latter observer, the diminution of acidity becomes perceptible about forty minutes after breakfast, and in the second hour after dinner; the secretion may be actually alkaline during the second or third hour after breakfast, remaining so about an hour; and during the third hour after dinner, remaining so for about three hours. The cause of the change is believed to be not so much the abstraction from the blood of acid to form gastric juice as the absorption into the blood of alkali produced by the digestion of the vegetable acids contained in the saline matters derived from the food. Consequently, it is especially likely to be well marked after meals consisting largely of fruits and of other substances in which salts of the vegetable acids are abundant. In medical practice the effect of food upon the reaction of the urine is seldom noticed except by accident, because what is passed from the bladder is generally a mixture of the secretions formed by the kidneys over a considerable period of time. But one must always be prepared to find a specimen alkaline, as for example, when it is passed in one's consulting-room in the course of the morning, by a patient who has been to the closet after his breakfast.

In some abnormal conditions, on the other hand, the urine passed throughout the whole twenty-four hours is found, when collected, to have an alkaline reaction. A distinction must now be drawn which is of the highest importance. The alkalinity may be due to putrefaction or, as it is termed, "ammoniacal fermentation" of the urine after it is secreted, but while it is still in the urinary passages. This change I shall describe further on; what characterises it is that the alkali is volatile, so that a red litmus paper turns blue when suspended above the urine, and not dipped into it, and that a paper turned blue by such urine regains its red colour when it is allowed to dry. On the other hand, urine which becomes alkaline after meals owes its alkalinity to fixed alkali, so that red litmus paper is affected only when dipped into it, and remains blue after it has dried. And so with the cases which I am about to mention, and in which the urine is found to be alkaline throughout the twenty-four hours independently of any putrefaction or fermentation in it. Quincke has observed this in patients with chronic vomiting, especially as the result of dilatation of the stomach; and he has doubtless rightly attributed it to the abstraction from the body of the acid of the gastric juice which normally should be reabsorbed into the blood. A like condition of the urine has also been noticed in patients who have the stomach regularly washed out. Under other circumstances a persistent alkalinity of the urine from fixed alkali is not common. But both Dr Bence Jones and Dr Roberts have observed such cases. Dr Roberts says that the patients were persons of debilitated constitution, anæmic after subacute rheumatism or gout, chlorotic, dyspeptic, or phthisical. Generally the urine would be alkaline for two or three days together, and then acid for a time, becoming again alkaline later on. But sometimes it remained steadily alkaline for weeks without intermission.

Urine which is alkaline from fixed alkali is commonly turbid when passed. It precipitates certain of its solid constituents, chiefly phosphates of the alkaline earths. The chief of them is the amorphous phosphate of lime ($Ca_3P_2O_8$) which collects as a flocculent deposit, always whitish and paler than the supernatant liquid, and thus distinguishable from the amorphous urates. It also forms an iridescent film upon the surface, the cause of which appears to be the escape of carbonic acid from the urine. Mixed with this is sometimes, though rarely, another phosphate of lime ($CaHPO_4 + 2H_2O$) which is crystalline. It forms rods or needles, smaller at one end, so as to be club- or bottle-shaped, and generally grouped together into stars, rosettes, fans, or sheaf-like bundles. Still more infrequent is the phosphate of magnesia ($Mg_3P_2O_8$) which forms elongated plates with oblique ends. It seems only to have been recognised by two observers, Tollens and C. Stein. It is, in fact, a very soluble salt, so that it is not likely to be precipitated unless there is a very large quantity of it: and, moreover, there must be no ammonia in the urine, since that base, if present, unites with the phosphate of magnesia to form another salt, the well-known "triple phosphate" ($MgNH_4PO_4+6HO_2$). This salt is not infrequently found in urine alkaline from fixed alkali, but in small amount as compared with its abundance in urine alkaline from ammoniacal fermentation, so that I shall postpone my description of it until I come to speak of that condition. All phosphatic deposits are instantly dissolved by acetic acid, which forms a test distinguishing them from other urinary precipitates. Carbonate of lime seems to be frequently mixed in small quantity with the amorphous phosphate of lime; sometimes it appears in small spheroids.

Urine, of which the reaction is not alkaline but neutral, or even faintly acid, sometimes throws down the crystallized phosphate of lime, and even the "triple phosphate" of ammonia and magnesia. A marked feature of such urine is, that when warmed in a test-tube it becomes cloudy and opaque, so that it may look exactly as if it contained albumen, owing to a separation of phosphates of lime that had hitherto been retained in solution in it. I shall have to advert to this point again when speaking of the tests for albumen. It has generally been attributed to the driving off of carbonic acid from the urine. But Salkowski has recently pointed out that this cannot be the case, since when the turbidity is but slight the urine often becomes quite clear again on cooling. Dr Walter Smith ('Dublin Jour. of Med. Sci.,' 1883) has since taken up this subject, and has arrived at the conclusion that the precipitation of phosphate of lime by heat depends upon "a nice adjustment of the proportions and basicity of the phosphatic salts in the urine." He imagines that when the fluid at the ordinary temperature contains the dicalcic phosphate ($Ca_2H_2P_2O_8$), held in solution by the presence of other salts, the effect of heat may be to resolve it into tricalcic phosphate ($Ca_3P_2O_8$) and monocalcic phosphate ($CaH_2P_2O_8$), the former of which is insoluble; cold, on the other hand, may lead to the inverse change and to a disappearance of the precipitate.

The presence of a phosphatic deposit in urine alkaline from fixed alkali may be regarded as a matter of no consequence, so far as concerns the formation of concretions within the urinary passages, since the amorphous phosphate of lime which forms the bulk of it has scarcely any tendency to cohere into solid masses. Only the very rare calculi which consist entirely of this substance can have had their origin in such a state of the urine. And there is no ground whatever for supposing the visible precipitation of phosphates, whether it be spontaneous or as the result of heat, to be an indication that these substances are being excreted in excess. To determine that, it would be absolutely necessary to make an exact quantitative analysis. Such analyses have been made in large numbers, but with the most meagre results, so far as their clinical value is concerned. There is therefore not even a *primâ facie* probability in favour of the view, which at one time was entertained by good observers, that a "phosphatic" state of urine is ever dependent upon an undue disintegration or waste of the nervous tissues, these tissues containing phosphorus as an important element. And certainly direct evidence in support of such a view is altogether wanting.

In the *treatment* of those cases in which the urine is habitually alkaline from fixed alkali the main thing is to improve the general health by tonics, change of air to the seaside or to a mountain health resort, exercise short of fatigue, and other measures tending to the same end. The direct administration of acids is found to have very little effect.

URINARY PIGMENTS.—With regard to the substances that give to urine its various shades of colour in health and in disease there is still much uncertainty and confusion. According to Salkowski and other recent writers, the principal urinary pigment is a substance to which Jaffé first gave the name of *urobilin* (= *hydrobilirubin*—Maly): it gives peculiar absorption lines in the spectrum, and a green fluorescence is given by its ammoniacal solution on the addition of chloride of zinc. Vierordt, however, has pointed out that this cannot be the only colouring matter, inasmuch as the spectrum of urine is not absolutely identical with that of a solution of urobilin. And Huppert, in his eighth edition of the well-known work of Neubauer and Vogel, declares that the real pigments of normal urine are still unknown. Urobilin, he states, is not present as such, but in the form of a "chromogen," which at once yields it on the addition of mineral acids. But in certain pathological conditions urobilin exists in the urine in a formed state; and the quantity that can be extracted is far greater than natural, though it still amounts to only from $\frac{1}{32}$ to $\frac{1}{16}$ in a thousand parts of the fluid. MacMunn finds the urobilin oxydised to choletelin. Urobilin ($C_{32}H_{44}N_4O_7$) can be derived from bile-pigment, or from hæmatin or hæmoglobin, by a process of deoxidation. Salkowski says that its origin within the human body is from the bile in the intestines, under the influence of hydrogen set free as the result of putrefaction and other chemical processes that take place there. In fact urobilin gives the fæces their colour, all the bilirubin which passes into the blood being converted into this substance.

It is especially in febrile urine, and in that passed by patients with obstruction of the venous circulation, that urobilin is found in excess. But of course it must be remembered that such urine is generally scanty, so that the increase may not be so great as it appears. Salkowski says that constipation seems not to augment the urobilin in the urine. In jaundice there is often a great excess, which, however, can be recognised only after the bile-pigment has been precipitated and removed. The urine may likewise be found loaded with urobilin before an attack of jaundice and after it has passed off. Surely its presence in jaundice (for which we have the authority of Hoppe Seyler) shows that absorption of deoxidised bile-pigment from the intestine is not the only source of this pigment in the healthy urine.

Indican.—The fact that indigo-blue is sometimes present in the urine was noticed many years ago by Prout; and afterwards by other observers, some of whom showed that this colouring matter in many cases made its appearance only when the urine had been exposed to the air. But it was Schunck, of Manchester, who first recognised in urine the constant presence of indican, a colourless material, which he had also discovered in plants and which readily passes into indigo-blue by oxidation. More recently, however, it has been found that the indican of urine is not identical with vegetable indican; according to Baumann, Briczar, and Tiemann, it is an indoxyl-sulphate of potass ($C_8H_6NKSO_4$). The only observations by which any clear light seems as yet to have been thrown upon the origin of this substance in the human body are those made by Jaffé, who observed in 1872 that indican could be made to appear in the urine of animals in large quantity by feeding them with a substance called indol, or by injecting that substance under the skin. Now indol is formed within the intestine in dogs, and to some extent in man, as the result of a change in albumen induced by the pancreatic ferment. The absorption of it from the intestine may therefore be fairly supposed to give rise to the presence of indican in the urine; and Jaffé has in fact detected indican in very large quantity in cases of obstruction of the small intestine or of strangulated hernia, and also in dogs after ligature of a loop of small intestine. But it is quite another question whether this is the sole, or even the usual, source of an excess of indican in the urine under morbid conditions. Senator ('Ctrblatt.,' 1877) and Heninge ('Deutsch. Arch.,' 1879) have recently investigated the conditions under which such an excess is met with. The former refers it especially to states of inanition and wasting, such as arise from cancer of the stomach, gastric ulcer, multiple lymphomata, phthisis with diarrhœa, granular disease of the kidneys. The latter insists that the excess is especially marked in cases in which wasting is dependent upon affections of the intestinal canal.

He observed it not only when there was constipation, but also when diarrhœa was present and even in cases of *cholera nostras.* On the other hand, in cases of catarrhal jaundice and in cases of cirrhosis of the liver, the amount of indican in the urine was always small. The general result of these observations appears to be that the recognition of indican in the urine is at present useless from a practical point of view. It is often found in large quantity in urine which is pale and contains little formed pigment. The test for indican, as given by Jaffé, is to add to the urine an equal volume of hydrochloric acid, and then to pour in drop by drop a solution of chloride of lime, shaking the fluid well, and adding no more of the chloride after a greenish colour begins to appear. If any considerable quantity of indican is present a blue colour will soon show itself; and if the quantity is very large indigo blue will be deposited in flocculi. A dilute solution of bromine may be used instead of the chloride of lime. The blue pigment may be afterwards extracted by agitating with chloroform or ether; and in this way the amount of it may be roughly estimated.

According to Brieger, a still more frequent constituent of human urine is an allied substance, skatoxylsulphate of potass, which is derived from *skatol,* this being (like indol) a product of the decomposition of albuminous substances within the intestine. I may mention that indol and skatol each have a strong fæcal odour. Jaffé's test with hydrochloric acid and chloride of lime gives a reddish-violet, instead of a blue colour, when the skatoxylsulphate is present.

Another urinary pigment which appears to be different from all of those hitherto mentioned, and of which the origin is as yet unknown, has been called uroerythrin. I shall presently speak of it as giving a pink or red colour to deposits of urates (p. 354). It is sometimes spoken of as identical with what Dr Golding Bird called "purpurin." Probably, however, he also included under that term several substances which are now described as distinct, since his test for it was to add hydrochloric acid to the hot urine, when he obtained a colour "varying from a delicate lilac to the deepest crimson."

In conclusion, it must never be forgotten that colours that may be mistaken for the effects of urinary pigments may be merely due to the administration of medicines. For example, rhubarb (says Dr Roberts) colours the urine a deep gamboge yellow, which is changed to red by the addition of ammonia. Senna communicates a brownish, and logwood a reddish, tinge. Santonin gives a conspicuous orange-yellow colour to the urine if alkaline, a rich golden-yellow if acid.

SALINE DEPOSITS.—Of some of the crystalline or amorphous materials that may be found as sediments in the urine, I have already had occasion to speak; for example, of tyrosine as a symptom of acute atrophy of the liver (p. 265), and of phosphate of lime in cases in which the urine is alkaline (p. 349). And the precipitation of the phosphate of ammonia and magnesia will be described further on, as an effect of ammoniacal fermentation. Under the present heading, therefore, I shall confine myself to giving an account of those deposits which require separate mention. It will be found that most of them possess clinical importance from one or the other, if not from both, of two different points of view; either as constituting the main indications of disturbance of the chemical processes occurring perhaps in some part of the body very remote from the kidneys, or else as involving the risk of the formation of gravel or calculus within the urinary organs themselves. They are (1) uric acid and the urates, (2) oxalate of lime, (3) cystine, (4) sulphate of lime.

Uric acid and the mixed urates.—In urine having the normal acid reaction uric acid exists in the form of acid salts of soda and other bases, which at the temperature of the body are fairly soluble. But as the fluid cools these are often precipitated. Very slight changes may disturb the balance between them and other saline ingredients, so as to separate the acid, which is then thrown down, inasmuch as it requires a very large quantity of water (14,000 parts of cold water) to hold it in solution. Scherer many years ago asserted that during the first few days after being voided, urine, as a rule, undergoes what he termed an "acid fermentation." This, however, is not now believed to be the case. By Voit and Hoffman ('Bayerisch. Acad. Sitzungsber.' ii, p. 279) it is maintained that the acid phosphate of soda (which is commonly held to be the cause of the acid reaction of urine) gradually takes away from the uric acid more and more of the bases with which it is combined. It is said that an appreciable diminution in the acidity of the urine may be produced in this way, inasmuch as the uric acid, being deposited in a solid form, is no longer capable of affecting the reaction.

Uric or lithic acid.—As a deposit from urine, this substance appears in the form of crystalline grains which have a reddish colour, so that they often look almost exactly like cayenne pepper. They commonly lie loose at the bottom of the fluid, but sometimes they adhere to the sides of a glass vessel, or may float in a film upon the surface. Their colour is not proper to the acid itself, for this, when derived from other sources, is colourless; it really belongs to urinary pigment, for which uric acid seems to have a strong attraction and which is consequently carried down with it. Dr Beale says that he has three or four times seen colourless crystals of uric acid deposited from urine which happened to contain hardly a trace of colouring matter.

The form of uric acid crystals is primarily that of a rhombic prism or lozenge, but they present a great many varieties of shape. Sometimes they are short and thick and barrel-shaped, or almost cubical; sometimes they form rods which seem to have rectangular extremities; sometimes they appear as flat plates which may be fiddle-shaped or halberd-shaped. Very often they form large stellate aggregations, and sometimes fan-shaped masses which may occasionally be connected together in pairs so as to have somewhat the character of "dumb-bells." Dr Ord has pointed out ('Med.-Chir. Trans.,' lviii) that the deviations in form which those crystals present from the regular four-sided or six-sided plates that are seen when the pure acid is crystallised from water, depend upon the presence of mucus and of colouring matter, which substances favour Rainey's "molecular coalescence" rather than crystallisation, so that by a kind of compromise the resulting crystals, instead of having sharp angles and straight sides, are more or less rounded off, and also tend to cohere together in masses having a common centre. He has also found that the presence of albumen in the urine still further modifies the form of the crystals, rendered them small and thick, with their angles more or less nearly equal, so that they may be said to be tub- or cask-shaped. The association with sugar, on the other hand, tends to produce flat and elongated crystals, which may have the typical hexagonal shape that is otherwise so rarely seen in specimens derived from urine. The microscopical character, or generally even the naked-eye appearance, of a deposit of uric acid is quite sufficient to distinguish it from any other substance that occurs in the urine. But unless the quantity be very small, the well-known murexide test can be easily applied.

Mixed amorphous urates.—The commonest of all urinary deposits consist of a loose pulverulent substance, which varies very much in tint, but is always of a deeper colour than the urine from which it is derived. It is often spoken of as brickdust-coloured, or "lateritious" (*later* = a brick). It generally settles quickly, leaving the urine above almost clear, but sometimes it remains a long while diffused through the fluid, especially (Dr Roberts says) when albumen is present. Not infrequently, if the urine has been put aside while still warm, different strata of the deposit in the same glass have different colours; they may be fawn-coloured, orange, brick-red, pink, or purplish. A part adheres to the side of the vessel as a sort of film or bloom, which is not very easily cleared away. With the microscope this precipitate is seen to consist of minute granules, which are coarser or finer, more or less opaque, according to the closeness of its aggregation. All doubt as to its nature may be removed by applying heat. As soon as the fluid is warmed it becomes bright and clear; and even when albumen is present there is seldom any difficulty in obtaining a satisfactory result, for the urates dissolve at a far lower temperature than that at which albumen coagulates, though of course more heat is required to clear up a dense precipitate from which the supernatant fluid has been poured off than if it were merely diffused through a bulk of urine.

Until lately the lateritious deposit was commonly spoken of in this country as consisting of the urate of ammonia, while German writers describe it as a urate of soda. In reality it consists of a mixture, in different proportions in different cases, of the urates of soda, potass, ammonia, and lime. Moreover, the quantity of uric acid in it is largely in excess of that which would correspond even with the acid salts of these bases, being in fact about twice as much, and making up 80 or 90 per cent of the whole precipitate. Dr Roberts says that this loosely combined acid can be separated from the acid lithates by warm water (which must of course be used sparingly), or even by cold water, with which the deposit is to be repeatedly washed upon a filter.

Crystalline lithates or urates.—It is a curious circumstance that urate of soda is never deposited from urine in those needle-shaped crystals with which we are so familiar in gouty concretions, and which may also be readily obtained artificially from solutions of the salt. In some cases, however, it forms opaque globular masses from which project spiny crystals, straight or curved. I have already alluded to these "hedgehog" or "thornapple" (*Stechapfel*) bodies as occurring in putrid urine. But they are also sometimes seen in patients (especially children) who are suffering from pyrexial disorders, and even (Dr Roberts says) in persons affected with gout. Its occurrence probably depends upon the urine being very scanty and concentrated, and being detained in the bladder. In one case a child three years old was suffering from fever, and had passed no water for three days. While Dr Roberts was examining the abdomen the child cried, and urine began to flow: the first that came was turbid and of a gamboge-yellow colour, containing the spiny masses; after about an ounce had passed there followed several ounces of clear fluid.

Clinically, deposits of uric acid and of urates have to be looked at from two points of view. There is, first, the question whether they are the cause, or likely to be the cause, of calculi, or of lumbar pain, hæmaturia, and other symptoms of pyelitis. Now, as regards free uric acid, much depends upon whether or not it is precipitated soon after the urine is passed from the bladder. As Dr Roberts remarks, if the crystals are seen before the urine cools, there is always a risk that they may also be found within the urinary passages. Even if the deposit takes place within three or four hours it is certainly not natural, though it hardly requires special treatment. But if it does not occur until after twelve hours or longer, it has no pathological significance whatever. Amorphous urates can never in themselves be of importance by producing irritation of the kidneys or bladder, since they appear never to be precipitated so long as the urine is of the temperature of the body. But the hedgehog crystals of urate of soda are thought by Dr Roberts to be not infrequently the starting-point of calculous formations.

Secondly, there is the question how far deposits of lithic acid or of lithates indicate a disturbance of the chemical processes by which in the blood or in various organs nitrogenised substances are prepared for excretion by the kidneys. Now, it cannot be too strongly insisted on that the formation of such precipitates is not in itself a proof that the quantity of uric acid is excessive. In the case of the amorphous urates a great deal depends upon the temperature to which the urine falls when it cools. It does not appear that there is any other cause than the lower temperature for the fact that deposits of these substances are so much more frequently observed in winter than in summer. Again, whatever diminishes the amount of water excreted by the kidneys increases the likelihood that urates will be precipitated. This seems to be the reason why amorphous urates are so often seen in healthy persons after violent exercise and after profuse sweating, and also in patients who are suffering from any disease, such as rheumatic fever, attended with perspiration in its whole course, or who are passing through the crisis of an attack of pneumonia when there is excessive perspiration. The degree of acidity of the urine is also of importance. Most observers say that urates are never deposited except from urine which is acid, but according to Salkowski the reaction may be neutral. In the case of free uric acid the degree of acidity of the urine is the most essential factor of all in determining its precipitation. Salkowski, however, states that it may occur even in urine which is neutral or alkaline, basing this assertion upon the observations of Voit and Hoffman, according to which the acid phosphate of soda may set free uric acid from the urates, even when it is present in such small quantity that it all becomes converted into the so-called neutral salt, which reacts like an alkali.

To determine the exact significance of deposits of uric acid or of urates would be of comparatively little consequence if it were easy to make quantitative analyses by which the amount of the acid in a given specimen of urine could be exactly ascertained. But unfortunately this is very far from being the case. The usual plan is to precipitate with hydrochloric acid, to collect the uric acid which falls down upon a filter, and to weigh it. This, however, is found not to give correct results, since a variable amount of the uric acid remains in solution; it may even happen that no precipitate occurs notwithstanding that uric acid is present. Salkowski, therefore, has proposed a method which depends upon the formation of a compound salt of sodium and silver; this, he says, is much more accurate and takes less time, but there are difficulties in carrying it out correctly. Dr Pavy has recently advocated ('Med.-Chir. Trans.,' lxii) the use of the ammoniated cupric solution, which he employs for the estimation of sugar. He finds that 0·01866 gramme of uric acid is required to decolourise 20 cc. of this test. The reducing action of healthy urine, however, is not due solely to the uric acid in it; a small quantity of sugar is also usually present, which takes part in the effect. Dr Pavy therefore repeats the experiment with a second specimen of urine from which all the uric acid has been precipitated by acetate of lead. The difference between the two results gives the amount of uric acid which the urine contains. Dr Pavy gives figures which appear to prove the accuracy of this method. The total quantity of uric acid excreted by healthy persons in the twenty-four hours is not large; as a rule, it is from three to eight grains. The proportion between the uric acid and the urea is usually as one to fifty or as one to sixty.

It was, I believe, Liebig who first suggested the idea that uric acid is formed by oxidation out of the same materials as urea, and represents a stage in the formation of the latter substance in which the oxidising process is as yet incomplete. Unfortunately this hypothesis seems to be capable neither of proof nor of disproof. But at least it fits in well with many important clinical facts. The most obvious way of testing it seems to be that of adding uric acid to the food, and determining whether or not the amount of urea excreted afterwards undergoes an increase. Wöhler and Frerichs originally proposed this experiment, and came to the conclusion that the uric acid was mainly converted into urea; other observers have since repeated it with a like result. Salkowski found in dogs that allantoin appeared also to be formed out of the acid, but as this must itself have arisen by a process of oxidation, he regards it as rather tending to confirm than to upset the view that uric acid constitutes a step in such a process. The same may be said likewise of the facts (to which I shall presently allude) which seem to show that oxaluric acid and oxalic acid may be formed in the body out of uric acid. And, on the other hand, it is a significant fact that in birds uric acid is derived from the same substances (aspartic acid, glycin, leucin, and asparagin) which in mammalia pass into urea. Urea itself, when given to birds, is said to undergo conversion into uric acid; this, at any rate, indicates how close is the relationship between the two bodies, though of course the change must be one of deoxidation and not of oxidation.

Clinically, several sets of facts have been adduced in support of the view that uric acid may be excreted in excess as the result of a deficient supply of oxygen, taking the place of urea, but this explanation seems to be open to question in every case. For example, as to the "uric-acid infarcts," which are seen in the kidneys of newly-born infants, Cohnheim remarks that he has observed them especially in strong healthy children who had breathed well, whereas they were often absent in cases in which there was pulmonary atelectasis, bronchitis, or bronchopneumonia. So, again, many observers have shown that there is in leuchæmia a marked increase of uric acid in the urine, both absolutely and relatively to the urea. This has been attributed to the deficiency of red discs as oxygen carriers. Pettenkofer and Voit, however, found ('Ztschft. f. Biol.,' v) that in a case in which the excess of uric acid amounted to 64 per cent., the absorption of oxygen, and the evolution of carbonic acid from the lungs were normal. Lastly, Bartels some years ago ('Deutsch. Arch.,' 1865) endeavoured to show that in various affections attended with insufficient aëration of the blood, the amount of uric acid in the urine is increased in proportion to that of the urea, the proportion rising from the normal rate of one to fifty or sixty up to that of one to thirty-five, or even of one to twenty-five. But experiments made on animals by Senator and others have for the most part failed to confirm these observations. And of late it has been shown by Fränkel, and since then by Fleischer ('Virch. Arch.,' 1882) that a constant effect of dyspnœa is actually to augment the secretion of urea. Possibly the over-activity of the respiratory muscles plays a part in bringing about this result, and at any rate we may, I think, agree with Salkowski that the conditions in dyspnœa are far too complicated to allow of our attributing solely to deficiency of supply of oxygen the changes in the urine that may be associated with it.

But it is especially in relation to disorders of the chylopoietic viscera that the meaning of deposits of uric acid or of urates has to be considered by practical physicians. Dr Roberts made during seven days a series of observations on a person who dined at 2 p.m. and afterwards took no solid food till the next morning. And he found that during the period when the urine was alkaline after the meal—which was from about 4 p.m. till 7 p.m. —the quantity of uric acid excreted in each hour was three times greater than it was from 9 p.m. till 11 p.m., or later on during sleep. The proportion which the acid bore to the rest of the urinary solids, and even to th water, was likewise greater, though, being alkaline, the urine of course threw down no deposit of urates. The food taken while these observations were being made was very simple. Consequently, it seems fair to conclude that the effect of rich food and of frequent meals in increasing the excretion of uric acid should be even more decided. And, as a matter of fact, many persons who habitually live plainly, and who ordinarily pass clear urine, find that the occurrence of a lateritious deposit is an inevitable consequence of any considerable indiscretion in diet; while other persons whose rule it is to indulge in the pleasures of the table secrete during a large part of every day urine that becomes turbid with urates as it cools. It is true that such deposits do not in themselves prove an excessive excretion of lithic acid, but if we also take Dr Roberts's observations into consideration, we may surely infer that this is the case. And, at any rate, no theoretical doubts can do away with the practical significance of this symptom, especially if it is associated (as we so constantly find it to be) with a foul tongue, yellow conjunctivæ, an irritable temper, and all the other signs which have been enumerated at p. 254 as symptoms of lithiasis. Of course, even if we admit that the amount of uric acid contained in the urine in such cases is absolutely increased, it does not at all follow that it is increased relatively to the urea. That substance itself may be increased likewise, though as it is readily soluble there is nothing to show it. But what seems to prove conclusively that deposits of urates are not merely an indication of excessive nitrogenous elimination as the result of the ingestion of undue quantities of nitrogenised food is that among the articles of diet which are most apt to be followed by the appearance of such deposits in the urine are some (such as sweet things, port wine and champagne) which are themselves non-nitrogenous. I do not see how it is possible to resist the conclusion that such substances are capable of disturbing the balance of the chemical changes which nitrogenous foods (and also probably the products of the waste of nitrogenous tissues) undergo in the liver or elsewhere in the body, and of causing an excess of uric acid to be formed. It is true that Dr Parkes says that no excessive excretion of uric acid occurs as the result of the experimental addition of either sugar or starch or alcohol to the food. But, in all probability, the limits of health were not overpassed in the observations upon which these statements were founded, as they are in the cases that I am referring to.

The presence of colouring matter and chromogens in the urine in excessive quantity is generally held to afford corroborative evidence of the same kind beyond that which is afforded by the mere presence of the uric acid or of the urates. I have already referred more than once to the attraction which urates exert not only upon urobilin but also upon uroerythrin. The deeper the tint of a lateritious deposit occurring in a non-febrile patient, the more surely would it be generally regarded as a proof of lithiasis. And I believe that the same conclusion may in the main be safely drawn from the formation of pigmentary matters in large quantity on the addition of mineral acids to urine. The late Dr Golding Bird, for example, was convinced that the presence of what he termed "purpurine"—detected by the violet colour which followed the addition of hydrochloric acid to the hot urine —was a proof of "derangement of the hepatic function." He thought it was sometimes a valuable clinical aid to diagnosis, in cases in which there was an abdominal tumour of doubtful nature, or in which it was uncertain whether ascites was due to hepatic disease or to chronic peritonitis. The same applies, I think, to the zone of pigment of various hues that is so commonly observed when nitric acid is poured through urine as a test for albumen. This seems to depend upon the presence, in different amounts in different cases, of all the substances mentioned at p. 351; the chromogen of urobilin, indican, and skatoxyl-sulphuric acid. And accordingly the tint is sometimes crimson, sometimes purple, sometimes bluish black. Perhaps the purple and indigo-blue colours may be sometimes merely evidences of constipation. A curious point, for the knowledge of which I am indebted to Dr Moxon, is, that in persons who are taking iodide of potassium the addition of nitric acid to the urine produces an orange-coloured zone, the appearance of which is quite peculiar.

Oxalate of lime.—This substance produces in the urine a cloud like that caused by mucus, and so little noticeable in an ordinary vessel that a patient's attention is very seldom, if ever, attracted by it. Dr Roberts, however, says that when the deposit is allowed to form in a conical glass vessel, it presents appearances which are quite characteristic. The sides of the glass become marked with fine lines, running transversely or obliquely, and making it look as if it were scratched; these are due to the crystallisation of the salt along minute irregularities left on the surface of the glass when wiped. In the urine itself there is near the bottom of the vessel a soft pale grey mucous-looking sediment, and above this a snow-white denser layer with an undulating but sharply-limited surface. If a drop of the deposit be taken up with a pipette and placed under the microscope, the oxalate is generally seen to form small octahedral crystals, beautifully transparent and lustrous, though in a case of jaundice they are said to have been found by Fürbinger stained yellow by bile. So transparent are these octahedra that all their facets and angles are commonly visible at the same time. They have a flattened shape, the principal axis being much shorter than the other two. In size they vary considerably; the late Dr Golding Bird found that the length of the sides of different specimens ranged from $\frac{1}{5600}$th to $\frac{1}{750}$th of an inch. They usually lie upon one pole, and then have the appearance which is aptly compared to that of a square envelope. But in different positions they may seem to have a rhombic outline, or even one which is rectangular or hexagonal. Other modifications in their shape are due to flattening of their lateral edges. This gives them a dodecahedral form, so that they consist of a rectangular prism with a pyramid on either summit. It is also said that half crystals are sometimes seen—pyramids on a square base. Much more rarely, oxalate of lime assumes a non-crystalline character; it is then generally said to appear in the form of "dumb-bells." Recent observers, however, have shown that the real shape of these bodies is that of a flattened rounded disc, with a central depression in each surface; it is when these discs lie on the side that they look like dumb-bells. They, too, vary in size. According to Dr Golding Bird the long diameter ranges from $\frac{1}{1420}$th to $\frac{1}{500}$th inch; their short diameter from $\frac{1}{2500}$th to $\frac{1}{750}$th of an inch. There was at one time some doubt as to the chemical nature of the dumb-bells. Dr Bird was disposed to think that they differed in composition from the octahedra, and suggested that they might consist of the oxalurate of lime. But this question has been finally set at rest by the observations of Dr Ord, who has shown that when oxalic acid and lime come into contact in the substance of a mass of gelatine, both forms are obtained. The assumption of a discoid, rather than of a crystalline character is doubtless due to the influence of colloid substances. Dr Beale has shown that dumb-bells are sometimes found in the interior of renal tube-casts. Chemically, oxalate of lime may be distinguished from all other urinary constituents with which it could be confounded by its being insoluble in acetic acid, but soluble in hydrochloric acid. The shape of the octahedra, however, is in itself a sufficient proof of their nature; it is only the dumb-bells which might be mistaken for like bodies of a different composition.

The origin of oxalate of lime is still unknown. There is no doubt that it may in part be derived directly from the food. Many vegetables and fruits contain this salt; not only rhubarb (which is so largely eaten in the spring in England) and sorrel (which on the Continent forms a common article of diet), but also, to a less extent, spinach, cauliflower, asparagus, tomatoes, apples, and grapes. Buchheim and Piotrowski are said to have found that from 10 to 14 per cent. of the quantity of oxalate ingested could be recovered in the urine. But Auerbach, detected oxalate of lime in the urine of dogs when fed entirely on animal food. Probably, therefore, oxalic acid may be formed within the human body also as the result of chemical changes. But as yet there is no certainty as to its source. English physicians of a former generation were inclined to think that it arose out of sugar. Its presence in the urine in a conspicuous form has sometimes been observed in connection with diabetes; but this can hardly be said to have much bearing on the matter. A more probable view is that it is derived from uric acid, by a process of oxidation with which chemists in the laboratory are familiar. Schunck's discovery that oxaluric acid in minute quantity is a normal urinary constituent is generally held to confirm this view, inasmuch as that substance forms a step in the process in question. But it seems not yet to have been positively shown that the ingestion of uric acid is followed by an increased excretion of oxalic acid.

But recent observations tend to show that what is clinically important in the case of oxalate of lime is, not its mere presence in the urine in greater or less quantity, but rather its precipitation in a visible form within the urinary passages. This was not the way in which Prout and Golding Bird regarded it. They, indeed, believed that its recognition was of the highest consequence, as affording a key to the right explanation and to the successful treatment of a group of symptoms which they enumerated, and of which the chief were a constant pain or sense of weight across the loins, irritability of the bladder, incapacity for exertion, impairment of sexual power, dryness of the palms of their hands and soles of the feet, a painful susceptibility to external impressions, nervousness, and an excitable temper, hypochondriacal depression, and emaciation. But in the urine of many patients who have such symptoms no oxalate can be found, while in that of many other patients who are quite free from symptoms it is present in abundance. Again, Fürbinger has shown that no conclusion as to the quantity of the salt contained in the urine can be drawn from the fact that crystals are discovered in the urine. Healthy persons, according to Schultzen ('Reichart's Archiv,' 1868), pass about a grain and a half in the whole of the twenty-four hours; in a morbid condition, in cases of jaundice, he found as much as seven and a half grains. But from what is known with regard to uric acid, it might well appear that this quantity is too small to allow of the possibility that the chemical changes concerned in its formation should be attended with any disturbance of the general health. And all observers are now agreed that the only clinical importance attaching to the presence of oxalate of lime in the urine is connected with the risk of its deposition in a solid form while the secretion is still within the body. Its significance, in fact, lies solely in its furnishing a clue to the probable presence of a calculus formed of the same substance, or to that of minute agglomerations producing lumbar pain, hæmaturia, or pyelitis. All of these effects will be discussed further on. At present I have only to consider what are the conditions under which such a deposition of the salt is apt to take place. Now, it has long been known that in some cases at least the octahedral crystals are formed slowly in the urine after it has been voided. They have been found to be both more numerous and larger in urine that has stood for a time than they were when it was first voided. Dr Rees even maintained in his 'Croonian Lectures' for 1856 that the oxalic acid itself arose out of uric acid in the urine itself, especially when heat was applied to it for testing purposes. This, however, seems to be very doubtful; and it may be noted that Neubauer ('Ztschrift. f. anal. Chem.,' 1868) found that even on adding oxalurate of ammonia and chloride of calcium to urine that no oxalate of lime was formed, the only change being the conversion of the oxalurate into carbonate of ammonia when the fluid putrefied and became alkaline. Moreover, Voit and Hoffmann seem to have satisfactorily accounted for the gradual separation of the oxalate of lime from urine which had for a time held it in solution. Neubauer, as far back as 1856, showed that this salt can be kept dissolved by a solution of the acid phosphate of soda. Now, it appears that when urine is allowed to stand the urate of soda in it becomes acted upon by the acid phosphate of soda so that an acid urate of soda, and ultimately free uric acid, are formed, while the phosphatic salt loses its acidity. Consequently, the conditions under which alone the oxalate of lime can be held in solution are no longer present, and it crystallises out. Obviously, therefore, the fact that octahedra are discovered in a patient's urine is in itself no reason for supposing that there is danger of the deposition of oxalate of lime in the renal pelvis or in the bladder. And it would seem to follow that the danger must be less in proportion as the urine is more highly acid. Almost all observers, however, are agreed that oxalate crystals are most often found in very acid urine; Salkowski alone, being influenced probably by theoretical considerations, speaks of them as being present chiefly in urine that is very faintly acid, neutral or faintly alkaline. They are often seen in association with deposits of urates or of uric acid. Radiating crystals of phosphate of lime also are not uncommonly present in urine containing oxalates. Dr Beale says that they occur in acid urine, a point about which I should have been unable to speak positively.

From what has been already stated it is evident that *treatment* is by no means necessary for all cases in which oxalate of lime is deposited from the urine. Dyspepsia and the effects of dyspepsia must be dealt with in exactly the same way as if no such deposit were present. As a rule the nitro-hydrochloric acid does more good than alkalies. Cold sponging, the use of a flesh-brush, exercise on horseback, change of air to the seaside or to an elevated health resort, are each of service. The food should be light, digestible, and varied. Leube, however, cites Cantani as having found that the urine was rendered free from the oxalate by an exclusive meat diet. Patients who have had lumbar pain or hæmaturia should be cautioned against eating rhubarb. To prevent the deposition of the salt within the urinary passages the best plan seems to be that of maintaining the naturally acid state of the urine; but Dr Roberts says that in some cases the urine has temporarily ceased to contain the crystals when it was rendered freely alkaline.

Cystine.—In 1805 Wollaston discovered in analysing a urinary calculus that it was composed of a peculiar substance, to which he gave the name of "cystic oxide." Subsequently the same substance, which is now almost always known as cystine, was found to occur as a light flocculent deposit from the urine. It looks very like a fawn-coloured sediment of amorphous urates, but with the microscope it is seen to be crystalline, consisting of hexagonal tablets, which Dr Roberts describes as having an iridescent mother-of-pearl lustre and as being often chased on the surface by lines of secondary crystallisation; there may also be thick rosettes of great brilliancy.

Acetic acid throws down from urine exhibiting this deposit an additional quantity of cystine, which had remained dissolved, and sometimes, perhaps, the acid might reveal the presence of it in urine which contained it in too small an amount to yield a spontaneous sediment. But the occurrence of cystine in any form is very rare; up to 1879 I believe that not so many as sixty instances of it had been recorded. Urine which deposits cystine is usually faintly acid, and is described as having "a honey-yellow colour, an oily appearance, and a peculiar sweetbriar odour." The deposit is instantly dissolved by caustic ammonia; when this evaporates, the hexagonal crystals reappear, but mixed with them there may also be highly refracting prisms lying singly or forming stars, such as are never seen in the urinary sediment itself.

The formula for cystine is $C_3H_7NSO_2$. From a chemical point of view the most remarkable fact about it is the large amount of sulphur which it contains, constitutes nearly 26 per cent. of its weight. One effect of this is that, when urine containing cystine decomposes, it evolves sulphuretted hydrogen, so that a glass vessel (if it happens to be standing in one) becomes blackened. Its composition forms an obvious point of resemblance between it and taurine, one of the elements of the bile. Hitherto, however, no observer has succeeded in making out that there is anything abnormal about the hepatic secretion in those persons who pass cystine in the urine. Nor has it been shown that this substance takes the place of other normal constituents of the urine containing sulphur. The amount of sulphuric acid, at any rate, appears not to be much less, if at all, in urine containing cystine than in that which is healthy. As to whether there is any change in the quantity of the organic sulphuretted compounds that exist in healthy urine there seems at present to be no evidence. The absolute weight of the cystine excreted daily is probably never very considerable; it is a light, though comparatively bulky, deposit.

It is a curious circumstance that cystine has several times been observed in the urine of persons related to one another as brothers or sisters. It is more often seen in males than females, and in children or young adults than in those who are older, but Dr Roberts had a patient whose age was fifty-seven. No condition of general ill-health is associated with the formation of this substance, and the only risk that appears to attach to it is that of the development of concretions or calculi. Some patients go on voiding it for many years continuously; in some cases it disappears from the urine for a time and then returns; ultimately the excretion of it may cease altogether. The treatment that has been advised for it consists in the administration of the nitro-hydrochloric acid, or of tincture of perchloride of iron, but there seems to be much doubt as to whether any benefit is to be derived from either of these remedies.

Sulphate of lime.—Considering that both sulphuric acid and lime are normal constituents of the urine, it is remarkable that the not very soluble compound which they form is so rarely deposited. As a matter of fact it has only been recognised in two instances, first by Valentiner ('Ctrlblatt.,' 1863) and afterwards by Fürbinger ('Deutsch. Arch.,' xx). It formed a bulky white sediment consisting of long needles and prisms with oblique ends, arranged in sheaves and rosettes. Valentiner's patient was an anæmic boy, Fürbinger's a wasted man affected with paraplegia. The conditions required for the production of such a precipitate appear to involve something beyond the mere presence of a moderate excess of sulphuric acid and of lime in a scanty urine, for in these respects there was no change in Fürbinger's case, when after about three weeks it gradually ceased to appear; and as the urine was highly acid he is inclined to think that there was a deficiency of the alkaline bases.

HÆMOGLOBINURIA.—In some cases in which the urine is reddened by the colouring matter of the blood, no red discs can be found in it. Within the last few years this condition has attracted much attention, and has been found to occur in various circumstances. It must be strictly distinguished from hæmaturia, in which blood itself is present.

Characters.—Urine containing hæmoglobin varies in its tint in different cases. It is sometimes of a pinkish hue, much more often dark red, chocolate brown, and occasionally almost black. It is often compared with strong tea or with porter. It may be clear and transparent when passed, but as it cools it is apt to throw down a thick sediment of lithates having a chocolate colour. Or the hæmoglobin itself may form a more or less abundant precipitate. With the microscope there is often only a granular *débris* to be seen, but in some cases a few shrivelled or altered blood-discs are visible. And sometimes the hæmoglobin itself appears in the form of rounded reddish-yellow drops, looking not unlike red discs, but variable in size and sometimes arranged in rows like the beads in a necklace. It may also be moulded into casts of the renal tubules, which have an opaque granular appearance and a reddish-brown colour. Sometimes, but rarely, it assumes the form of crystals. Hyaline casts in small numbers are said to be also present in some cases, and oxalate of lime crystals, the occurrence of which, however, is probably altogether accidental.

When urine containing hæmoglobin is heated, it yields a coagulum which has a brownish-red colour, and is described as differing from the ordinary coagulum of albuminuria in floating upon the surface of the fluid, instead of subsiding quickly to the bottom. This coagulum is said to be formed solely by the albuminous part of the hæmoglobin itself, no serum-albumen being present. Cohnheim speaks of a case in which Roux found that there was enough iron in the urine to correspond, according to that view, with all the albumen. If there is any doubt as to the nature of the colouring matter, the various chemical tests may be applied that I shall describe when speaking of hæmaturia. With the spectroscope a perfectly characteristic appearance is obtained. This consists in the presence not only of the well-known absorption bands of oxyhæmoglobin in the yellow and in the green between the solar lines D and E, but also of a third broad band in the red between the lines C and D but nearer to C. This third band belongs to a modification of hæmoglobin, which is called methæmoglobin, and which is intermediate as regards the amount of oxygen combined with it between oxyhæmoglobin and reduced hæmoglobin. It is probable that the change into methæmoglobin occurs in the blood before its excretion by the kidneys, inasmuch as Marchand has shown that in poisoning by chlorate of potass (which we shall find to be one of the causes of hæmoglobinuria) the blood itself yields a spectrum showing the band between C and D. When decomposition occurs in urine, any methæmoglobin it contains is converted into hæmoglobin.

Ætiology.—Hæmoglobinuria is probably always the result of a disintegration of red discs within the circulating fluid, or at least of the escape of the hæmoglobin from their "stroma." It is not known to be ever due to any primary alteration in the structure, nor to any perversion of the functions of the kidneys. The causes of the blood change are various. One of the simplest of them is the transfusion of blood from another kind of animal—as when, for example, lamb's blood is thrown into the veins of a dog—the explanation being apparently that the foreign red discs are broken up by the blood-serum of the animal into which they are introduced. Heat, again, may destroy the vitality of the corpuscular elements of the blood. Cohnheim says that hemoglobinuria is a very common symptom after extensive burns, if they are not too rapidly fatal, though I believe that this has hitherto attracted less notice from surgeons than from pathologists who have made experiments upon animals. As a result of heat-stroke, the affection is said to have been observed in Germany. It has been described as a complication of enteric fever (during a relapse) by Immermann ('Deutsch. Arch.,' xii), of scarlet fever (on the twentieth day) by Heubner

('Deutsch. Arch.,' xxiii), and of ague by Stolnikow ('Petersburg. med. Woch.,' 1880).

In other cases hæmoglobinuria is produced by the action of some poison absorbed into the blood; among the substances which have been known to cause it, either in animals or in man, may be mentioned hydrochloric acid, sulphuric acid, chlorate of potass, nitro-benzol, naphthal, and carbolic acid. Eitner has recorded a fatal case ('Berl. klin. Woch.,' 1880) in which it was set up by the inunction of pyrogallic acid into the skin of a patient affected with severe psoriasis. In several instances it has been due to the entrance of arseniuretted hydrogen gas into the air-passages.

The *symptoms* which accompany hæmoglobinuria from these various causes differ in intensity in different instances, but they seem always to belong to a common type. In severe cases the patient is perhaps seized with a rigor; vomiting and diarrhœa then set in; he becomes collapsed and cyanosed, falls into a state of stupor, and dies. From a clinical point of view, chlorate of potass far surpasses in importance all the other toxic causes of the affection. Hofmeier has collected ('Deutschr. med. Woch.,' 1880) no fewer than twenty-seven cases, all but four of which proved fatal. Some of the patients took the salt by mistake for sulphate of soda or for some other saline aperient. But most of them had it regularly prescribed for them, the mischief having arisen either from the dose being unduly large or too frequently repeated, or else from the swallowing of large quantities of a solution intended only as a gargle. In young children it would appear that from one to two drachms in the course of twenty-four hours is a dangerous quantity; in adults perhaps from two or three drachms upwards. In many instances the disease for which the chlorate of potass has been ordered has been diphtheria, and one cannot help suspecting that in a large number of other cases the salt may have produced like effects without their real cause having been suspected, the state of the urine having been attributed to a diphtheritic nephritis, and the severe constitutional symptoms having been regarded as indicative of the "collapse" which sometimes proves so rapidly fatal in diphtheria.

Anatomy.—The post-mortem appearances afford a ground of distinction. In all fatal forms of hæmoglobinuria the kidneys are found of a deep chocolate colour, presenting radiating lines and striæ, of which the tint is even darker than elsewhere. With the microscope the renal tubes are found to be completely plugged with masses of hæmoglobin. These have also been shown by Dr Bridges Adams to be present in the Malpighian capsules—a point of some importance as showing that the hæmoglobin is excreted through the tufts rather than the epithelium of the tubes. In one instance Hofmeier further noticed that the spleen had a peculiar reddish-brown appearance on section, and that the medulla of the femur in its upper half was brown. Ecchymoses have also been present upon the mucous membrane of the stomach and intestines.

The only other toxic cause of hæmoglobinuria to which I need specially allude is arseniuretted hydrogen. Eitner has recorded ('Berlin. klin. Woch.,' 1880) four cases due to this poison, in which the sufferers were a professor of Physics and three of his students. The professor had two attacks of hæmoglobinuria, separated by an interval of some days, and resembling the paroxysmal form of the affection in all respects except perhaps in having a rather longer duration. It was not until other persons were found to have suffered from like symptoms that their real nature was suspected. He and his pupils had, in fact, been repeating Tyndall's experiment of inhaling hydrogen gas, for the purpose of showing that the pitch of the voice becomes altered by it; and the zinc used in generating the hydrogen was impregnated with arsenic.

Spontaneous hæmoglobinuria occurs in circumstances which vary somewhat in different cases. Winckel has related ('Deutsch. med. Woch.,' 1879) a most remarkable outbreak, which occurred in 1879, in the lying-in institution at Dresden, where, between March 20th and April 29th, twenty-four newly-born infants were attacked by it, of whom twenty-three died. The symptoms were in every instance similar; the child, generally about the fourth day after its birth, became suddenly cyanosed and collapsed, with cold extremities, and it succumbed on an average within thirty-six hours. There was seldom any diarrhœa or vomiting, but the skin had in many cases an icteric tinge. The respiration and the pulse were very rapid; the temperature in the rectum was 99° or 100°. At many points the superficial veins became visible as dark thin lines, and when they were incised no blood escaped, but on pressure a brownish-black fluid of syrupy consistence could be squeezed out. The urine was of a brown colour, and exhibited the usual characters of hæmoglobinuria. Death was preceded by convulsions. On post-mortem examination the kidneys were dark brown and their papillæ showed plugs of hæmoglobin. The chief other morbid appearance was an enlargement of the mesenteric glands and of the spleen, the latter being tough and of a brownish-red colour. Every effort was made to discover the cause of this affection, but without success. Winckel proposes to term it "*Cyanosis infantilis icterica perniciosa cum hœmoglobinuria.*" There can, I think, be no doubt that its essential character was an intense and rapid disintegration of blood-discs within the circulating fluid. But why this should have occurred in a number of young infants in succession, themselves apparently healthy at birth, the offspring of healthy mothers, and in a hospital where no like disease had been observed before, remains a mystery.

Paroxysmal hœmoglobinuria.—In marked contrast with this fatal infantile form of hæmoglobinuria is one to which adults are chiefly liable, and which repeats itself over and over again without any danger to life and often with but trifling disturbance of health. It was first fully described by Dr George Harley, in the 'Med.-Chir. Trans.' for 1865, but a case of it may be found recorded by Dessler in 'Virchow's Archiv.' for 1854. Dr Harley called it "intermittent hæmaturia," and it has since been commonly known as "intermittent hæmatinuria" (Gull, 'Guy's Hosp. Rep.,' 1866), but the best name for it appears to be paroxysmal hæmoglobinuria.

In some few instances no cause for it can be discovered. I have seen two such cases, which occurred during the height of summer, in June, 1876, and in August, 1880. In neither case were any subjective sensations, except a pain in the loins, associated with the discharge of urine which was almost black. Fleischer has recorded ('Berlin klin. Woch.,' 1881) an instance in which a soldier was attacked every time he marched for an hour or two, although no other kinds of bodily exercise had a similar effect. But in the immense majority of cases the affection is due to one particular exciting cause, namely, *cold.* Sometimes the degree of cold that causes an attack (especially the first attack) is notably excessive, or, at any rate, far beyond that to which the individual is accustomed. A London physician, a friend of mine, observed hæmoglobinuria for the first time when he was one day fishing in Scotland in a biting wind, and his next seizure occurred some months later while he was skating without a great coat. But he subsequently became liable to the affection when the provocation appeared inadequate to produce such a result, as, for example, after standing four or five minutes at a railway station on a foggy morning, or riding two or three miles in a hansom cab. And in some instances it is stated that all but the early attacks have been altogether independent of cold. A patient who was seen by Dr Roberts said that he was just as bad in the summer as in the winter. As a rule, however, the affection ceases entirely during the warm part of the year, returning in the cold season, perhaps for many years in succession. That this is entirely a question of temperature has been shown by Rosenbach, who, by means of a cold foot-bath, brought on an attack during the summer in a person liable to the disease. Dr Roberts relates two cases, in each of which there were as many as three attacks in the day for days together. But the rule is that they recur at much longer intervals and generally quite irregularly. The part of the day at which they are most apt to take place is the *morning.* This circumstance is doubtless related to the fact that the temperature of the body is naturally lower then than it is in the afternoon and in the evening. It is especially when the patient is exposed to cold directly after breakfast, before the meal has been digested, that hæmoglobinuria is apt to show itself; and a cupful of warm beef-tea on first waking may prove an effectual preventive. Mental or bodily exhaustion, as from sexual intercourse, or from study late at night, favours the occurrence of an attack. And a like influence has appeared to me to be exerted by the free use of wine at dinner, probably as a result of the recoil from its stimulant action as well as of its relaxing the cutaneous vessels. Anxiety and nervousness also play a part in the ætiology of the affection in some cases. It is seen particularly in persons whose hands and fingers are liable to turn cold on excitement, and in those who (themselves or their relations) are liable to *digiti mortui.* The previous occurrence of *ague* has been noted in some instances, and it seems not impossible that this may sometimes be a more or less direct cause of hæmoglobinuria just as it is of splenic leuchæmia. The only doubt is whether the peculiar appearance of the urine may not have been overlooked during the early attacks, and whether they may not therefore have been wrongly ascribed to the influence of malaria, especially in countries where ague is prevalent. This doubt would of course be set at rest if there were a clear history of tertian or quartian periodicity. Syphilis was present in a case observed by Ehrlich; and a patient who was under my care in the clinical ward of Guy's Hospital in 1882, and who did not improve at all under the usual remedies, was subsequently cured by Dr Moxon with mercurial treatment; in that instance the spleen was much enlarged.

The disease is very much more common in men than in women. I have, however, seen one case in a female. It is most apt to occur in young adults, and up to the age of forty or fifty, but it is said to have been observed in a child only two years old. Dr Druitt, to whose case I shall presently refer, was fifty-one or fifty-two when he was first attacked.

The *symptoms* of paroxysmal hæmoglobinuria vary in different cases. One of the most constant is a feeling of languor and weariness, with a disposition to yawn again and again. With this there may be a feeling of chilliness, so that the patient is inclined to huddle up to the fire. The fingers and the toes—some or all of them—may turn white and dead. The most graphic account of the disease that I know of is in one that was given by the late Dr Druitt in the 'Med. Times and Gazette' for 1873; it is now, I suppose, an open secret that he himself was the patient. He describes his palms and his soles as becoming "cold, wet, blue and cramped, like those of a cholera patient." At other times he would have numbness of the right foot and of the left hand, without coldness. Or his nose, or some part of his cheek, would become first pale, then red, then purplish, and at last quite black. I have seen several instances in which the ears not only turned livid during the seizures, but failed to regain their natural appearance afterwards, so that a reddish-brown eschar formed along the edge of the helix, leading to a permanent loss of its substance.

While these symptoms are developing themselves, and until they have entirely passed off and been succeeded by a feeling of warmth and comfort over the whole body, there may be nothing to show that the secretion of urine is other than normal. There is often no desire to micturate. In a case that I have had many opportunities of watching it has sometimes been only after the lapse of several hours that any water has been passed, when the patient has almost forgotten that he had been chilly; so that he has been quite surprised to find it like chocolate or porter. But in other cases the bladder is irritable, and Dr Druitt says that he on some occasions suffered from great pain in the bladder, and was obliged every half hour to void urine, which then was peculiar in being of a bright scarlet colour. Sometimes, but not always, there is pain in the back, or radiating across the abdomen or down the thighs. Retraction of the testes also is mentioned by Dr Roberts.

The bodily temperature during a seizure of paroxysmal hæmoglobinuria is, I believe, always normal. Dr Druitt says that during the course of his illness (which had then lasted six years) he had severe attacks of remittent pyrexia, in which the thermometer would rise to about 103° in the evening, and fall to 100° in the morning; jaundice was always present, but the urine during these periods never contained any blood-colouring matter. So far as I know the pulse-rate remains in most cases unaltered; Dr Druitt, however, says that his pulse generally fell to 55 or 50 when the hæmoglobinuria was about to occur.

One of the most remarkable features of the disease is the rapidity with which, after a seizure, the urine regains its normal characters. The patient may once or twice, or oftener, have voided a fluid like porter; and that which he passes an hour or two later is perfectly clear and pale. Even in Dr Roberts's cases, in which there were two or three attacks in the course of a single day, colourless urine was secreted in the intervals. It is also curious how completely the appetite is unaffected; a patient who has had a well-marked attack in the morning may at one o'clock eat a hearty dinner as usual, sometimes even before he has discharged from his bladder the black urine formed during the attack.

It has now been well ascertained that the starting-point of paroxysmal hæmoglobinuria (as of other forms of the affection) is a disintegration of red discs within the circulating fluid. A microscopical examination of the blood does not, indeed, show any marked change in it, as I myself have found, and as has been stated also by other observers. Küssner, however, took blood with a cupping glass ('Deutsch. med. Woch.,' 1879) from a patient on six occasions during a seizure, and each time found that the serum was of a ruby-red colour, whereas at other periods it had the normal yellowish appearance. A still more striking experiment has since been performed by Ehrlich ('Deutsch. med. Woch.,' 1881). Having under observation a woman who was liable to the disease, he bound an elastic ligature round one of her fingers, and placed it for a quarter of an hour in ice-cold water, and afterwards for the same length of time in tepid water. Such a procedure on a healthy person produces no change in the blood, but in the patient in question it caused the red discs to break down in considerable numbers. When a drop of blood from the finger was placed in a capillary tube and allowed to coagulate there, the serum was distinctly seen to be reddened. And with the microscope the blood was found to contain "phantom" red discs or stromata (*œcoids*) of discs that had completely or partially lost their hæmoglobin, though there were also many normal discs, as well as pœcilocytes and microcytes. Evidently, therefore, the disease essentially consists in an undue sensitiveness to cold on the part of certain of the red discs of the blood.

In some hæmoglobinuric patients, however, there may be observed a circumstance which at first sight seems inconsistent with this view of the disease. It is that attacks of chilliness which fall slightly short of the degree of severity necessary to bring about an excretion of hæmoglobin, render the urine albuminous. This is a fact which I have verified on many occasions, the patient being perfectly free at all other times from any indication of Bright's disease. I have generally been inclined to account for it on the hypothesis that the albuminuria is really an altogether independent affection, resembling other forms of transitory albuminuria that I shall have to describe in a subsequent chapter. But perhaps the true explanation is that when the disintegration of red discs occurs only to a moderate extent all the hæmoglobin which is set free splits up within the blood into globulin and hæmatin, as it is well known to do under other circumstances. The albumen (or globulin) may then be fairly supposed to be incapable of remaining as a constituent of the serum, and to be excreted as such by the kidneys. It is, indeed, certain that even in ordinary cases of hæmoglobinuria a part of the hæmoglobin is decomposed so as to form a pigment resembling that of bile, if not altogether identical with it. The proof of this is the sallow bilious appearance which is invariably presented by patients who frequently suffer from the disease, and which is sometimes apparent even after a single severe attack.

Paroxysmal hæmoglobinuria has never been known to destroy life, nor does it appear to entail any serious consequences, though one can hardly doubt that if the attacks were allowed to recur frequently and for a great length of time the patient's health would ultimately break down. I have once seen chronic Bright's disease in a man who had previously suffered from hæmoglobinuria, and had lost the margins of his ears from it. The attacks may continue for many years in succession, or they may cease after a few weeks or months. Dr Druitt had suffered for six years when he published the account of his case; in another recorded instance the disease ran on for eleven years.

In the *treatment* of an attack the essential thing is warmth. If necessary the patient may be put to bed. Probably the best thing to drink is a basin of hot soup; I should not myself recommend brandy and water. When there is severe pain in the back it may be relieved by the application of mustard, or by dry cupping, or (Dr Druitt says) by the internal administration of extract of hyoscyamus.

But the important thing is to prevent the recurrence of the disease. The utmost care should be taken to avoid exposure to cold in the early part of the day, and in such circumstances as may be shown by experience to be injurious in the particular case under observation. The meals should be so arranged that there may be no deficiency of already digested food in the body at the time when a cold journey is to be made, or when any unaccustomed task is to be gone through that may cause nervousness and anxiety. The clothing must be warm; fur-lined boots and gloves are not to be despised; flannel drawers and a wash-leather jerkin should be thought of. Dr Druitt found a mustard bath useful, and he speaks highly of other forms of warm bath. But I should think it a mistake to use measures by which the superficial capillaries are in the first instance dilated. I have often noticed when I have come home on a cold day that after washing the hands with hot water the fingers have turned quite white, whereas up to that time they had shown no unusual appearance. And Dr Druitt himself says that his legs more than once became suddenly white when plunged into hot water (or directly afterwards ?).

The question of removal to a hot climate during the winter should in severe cases be seriously considered; Dr Druitt himself went to Madras, where he escaped the disease almost entirely. During the summer a bracing air is probably advisable.

Throughout the twenty-four hours the habits should be so arranged that the body shall become as little exhausted as possible. The patient should not spend his evenings in heated rooms, nor should he devote them to arduous mental work, but should go to bed early. He should have nutritious food in the latter part of the day, but little or no alcohol; for its effect is an immediate stimulation that rapidly passes off.

The one medicine that seems to have a marked effect in warding off the attacks is *quinine*. It must be given in full doses. It sometimes proves perfectly successful, so that the patient becomes again able to live his usual life without fear of the disease. But in severe cases, like that of Dr Druitt, no permanent benefit can be attained by it, even when it is used in such quantities as to cause singing in the ears and other disagreeable effects. Salicine, the tincture of iron, and arsenic, may each be of service in their turn. Chloride of ammonium is said to have done good in one instance. If syphilis is present, iodide of potassium or the bichloride of mercury may work wonders. Probably it is bad practice to prescribe mercurials or other active purgatives on account of the sallow icteroid complexion of the patient; for this is really not due to liver disorder but to quite a different cause.

PUTREFACTION OF URINE. ALKALINE OR AMMONIACAL FERMENTATION. —As is well known, urine left exposed to the air soon undergoes a change which renders it turbid and offensive to the smell, and which may be regarded as putrefactive. It is of some practical importance to the physician to be aware that this change takes place earlier in hot than in cold weather, that it is accelerated by the presence of pus or blood or mucus, and still more by admixture with even the smallest quantity of urine already putrid, and that it occurs much less rapidly in urine which is concentrated and highly acid than in that which is pale and watery and of a faintly acid, or neutral, or alkaline reaction. But infinitely more important is the fact that in certain circumstances it may take place within the urinary passages themselves, inasmuch as it is then a fertile source of inflammation, setting up cystitis, pyelitis, and even nephritis.

Putrid urine may be recognised by its penetrating fœtid odour, an odour which is often unfairly termed urinous (since it is quite unlike that of healthy urine), and the immediate cause of which has not been exactly ascertained. It also gives off carbonate of ammonia, the pungency of which may irritate not only the nose but the conjunctivæ, if the vessel containing the urine be held close to the face. This carbonate of ammonia arises from the decomposition of the urea. It gives to the urine an alkaline reaction, consequently the change which produces it is often spoken of as the "alkaline" or the "ammoniacal fermentation." That the alkaline reaction is due to this cause and not (as in other cases) to the presence of fixed alkali, can of course be easily determined. A piece of reddened litmus paper, or of turmeric paper, may be suspended in the mouth of a covered vessel over the urine but without touching it, when it will be found slowly to change its colour. Or the paper, after being dipped in the urine, may be left exposed to the air to dry, in which case the change in its colour that at first occurred will presently disappear. It must be remembered, however, that urine which was originally alkaline from fixed alkali may afterwards become putrid: in that case it will change the colour of paper suspended over it, but nevertheless paper dipped in it will continue to show an alkaline reaction after being dried.

Another character of urine that has undergone this change is its turbidity, a condition which filtering will not remove. The microscope shows that the cause of it is the presence of innumerable bacteria. It is by these bacteria—*micrococcus ureæ,* occurring singly, or two or more in chains, or in a group with mucus zooglœa—that the ammoniacal fermentation is brought about, probably immediately by a ferment which they secrete. Urine placed in clean vessels, and guarded from the approach of organisms from without, can be kept free from this change for an indefinite length of time: and so, also, it is the case within the living body. Some observers have maintained, however, that mere stagnation of the urine in the bladder, or in the pelvis of a kidney, suffices to render it putrid, especially if it contain mucus or cast-off epithelium. But that this is not the case has been proved by experiments on animals, in which the neck of the bladder or ureter has been ligatured, since the urine has then remained acid. In too many cases, the surgeon himself has introduced the bacteria into the bladder by using catheters or other instruments which had not been cleaned with the scrupulous care necessary to render them "antiseptic." This danger was, I believe, first pointed out by Traube, in 1864. The case which drew his attention to it was that of a man who had apparently had a distended bladder for two years, but whose urine was clear and acid when it was first drawn off by a catheter at the end of that time, whereas the next day it was turbid, and within six days it became alkaline, ammoniacal, and slightly fœtid. It is, however, the fact that stagnation of the urine in the bladder favours the occurrence of putrefaction in it. Cohnheim says that fluid containing bacteria may actually be injected into the bladder of a healthy dog without ill-effects, because they are all expelled the next time that the animal micturates. And in men, when this change has once occurred in the urine, nothing tends so much to keep it up as an inability on the part of the bladder thoroughly to empty itself, so that some of the putrid fluid is always left behind, and this induces the putrefactive process in that which is afterwards secreted. It is a notorious fact, too, that formerly, when catheterism was constantly practised without any precautions against the introduction of septic matters, the urine seldom became ammoniacal unless either the bladder was paralysed or the urethra in some way obstructed. In some cases, however, the putrefactive change takes place in the urine when no instrument has been passed. The bacteria may then have found their way along the urethra from outside, possibly through a layer of mucus which may have been left upon the surface of the mucous membrane in the act of micturition, when the urine contains much mucus. Or otherwise they have come from the blood, having passed out through the renal glomeruli with the urine. Lastly, in some rare instances an abscess has opened into some part of the urinary passages, and brought bacteria with the pus.

One effect of the occurrence of ammoniacal fermentation, whether in the body or out of it, is entirely to alter the conditions under which the solid ingredients of the urine are held in solution. The phosphates, in particular, are precipitated. The phosphate of lime ($Ca_3P_2O_8$) comes down in the form of granules; never, so far as I know, in the dumb-bells which appear under some other circumstances. Nor, again, does the crystalline phosphate of lime seem to occur in ammoniacal urine. But there are always brilliant crystals of another salt, the phosphate of magnesia and ammonia ($MgNH_4Po_4 + 6H_2O$), commonly spoken of as "triple phosphate." The form of these crystals is that of a triangular prism with bevelled ends; but they are liable to a great many modifications by planing off of their edges and angles, and sometimes their sides become hollowed out. Some of the prisms may be so short that their bevelled ends meet one another on one edge; they then look not unlike the octahedra of oxalate of lime. The addition of acetic acid at once distinguishes them; it dissolves all phosphatic deposits. Octahedra of the oxalate of lime, however, are sometimes present as well. Another element of the precipitate in putrid urine is formed by the urates of soda and ammonia; the former appears as round or irregular masses, from which project spiny crystals, straight or curved; the latter as opaque globular bodies, or as very small, slender dumb-bells, which may lie athwart one another so as to form a cross, or be united into a rosette. It is to be observed that the precipitate consisting of these various substances has no tendency to carry with it urinary pigments, like that of the amorphous urates which occur in acid urine; it therefore has usually a white colour, though Dr Roberts says he has known the urate of ammonia in putrid urine to possess a beautiful violet hue. Besides the deposit, there is often on the surface of the urine an iridescent film containing the same elements. An important character of the mixed phosphates and urates thrown down as the result of the ammoniacal fermentation is their liability to undergo concretion into a mortar-like mass; this forms a large part of many calculi, and it may also collect upon the end of a catheter left in the bladder, and upon the surface of the mucous membrane itself.

It is often of great consequence to determine whether the occurrence of the alkaline fermentation is limited to the urine which has already entered the bladder, or whether the bacteria which produce it have also made their way up into the ureters and into the renal pelves. Sometimes this may be made out by very carefully washing out the vesical cavity, and by drawing off a few minutes later the first few drops of urine that collect. If this is found to be acid the change takes place only in the bladder. How quickly the reaction may be reversed is well shown when there is extroversion of the bladder, so that the orifices of the ureters are exposed to view. In a case of this kind Dr Rees many years ago found (Croonian Lectures on Calculous Disease, 1856) that the urine as it flowed out from the orifices possessed its natural amount of acidity; but when reddened litmus paper was applied a quarter of an inch lower down, so as to test the urine after it had passed over that short distance of mucous membrane, its blue colour was restored. This result, however, is attributed not to an ammoniacal fermentation (for which there was not sufficient time), but to the admixture of alkaline exudation, from the reddened and inflamed surface.

In ordinary cases, before putrefaction of the urine within the bladder begins, cystitis is often already present. And even when that is not so, its occurrence is rarely long delayed. Urine which has undergone this change acts as a powerful irritant, though there seems to be some doubt whether the inflammation is set up directly by the bacteria, or rather by the carbonate of ammonia that results from their presence. The exudation of a large quantity of pus follows. This, however, does not retain its usual characters, but is converted by the ammonia into a viscid, semi-transparent, mucoid material, which often blocks the urethra, and causes the patient severe pain. In a vessel, it collects at the bottom, and coheres so firmly that when the urine is poured out it does not separate, but glides away as a ropy mass. The cystitis is often intense, leading to diphtheritic infiltration and sloughing of large tracts of mucous membrane (see vol. i, p. 57). And the inflammatory process often extends up along the ureters, and reaches the kidneys, as will be pointed out further on.

In this way ammoniacal fermentation of the urine often becomes indirectly the cause of death. It is therefore a matter of the highest importance to prevent its occurrence, and to check it whenever it shows itself. The strictest antiseptic precautions should be observed whenever a catheter is introduced, or when any other operation is performed upon the urinary organs. And if the change has already taken place before the patient comes under observation, the bladder should be washed out regularly with warm water containing borax and boracic acid. Dr Roberts says that the urine may sometimes be restored to its natural condition by making the patient drink large quantities of fluid at regulated intervals, so as to keep the bladder washed out by the renal secretion.

It would seem that an ammoniacal state of the urine, at the time when it is voided from the bladder, is not in itself a proof of the occurrence of putrefaction in it. At least, Dr Roberts says that he has observed this in two cases of advanced Bright's disease, without there being any evidence of delay in evacuation, and without any part of the urinary passages being afterwards found inflamed at the autopsy. He does not mention whether bacteria were present. Graves gives two cases, in each of which the urine, although free from any smell of putrescence, contained a large quantity of carbonate of ammonia, even when it was passed soon after being secreted. In one (a case of dropsy) the fluid was tested for urea, which was found absent; the other was a case of fever.

Non-septic Bacilluria of Roberts.—The presence of bacteria in the urine is not in itself proof that the ammoniacal fermentation has taken place, or is about to take place in it. Dr Roberts, in the 'Transactions of the International Medical Congress' held in London in 1881 (p. 157), has related several cases, in which the urine when voided was opalescent, and full of micrococci and moving rods. In two of the cases it had a disagreeable odour. But it was acid in reaction, and showed no greater (and perhaps a less) tendency to decomposition than healthy urine. Moreover, in the course of about twenty-four hours, the bacteria sank slowly to the bottom, leaving the liquid itself clear, and it remained in that condition and retained its acidity for several days afterwards. The organisms seemed to be incapable of multiplying in the urine, even when it was kept at blood-heat. Dr Roberts was therefore inclined to think that their original seat of development was in some part of the mucous membrane of the urinary tract. Some of the patients had had catheters introduced at a former period of their lives; in others this had not been the case. Two only were women, and one of them had no symptoms beyond a slightly increased frequency of micturition, especially at night. The others were men, and they suffered from scalding pain in passing water, with frequent desire to micturate, symptoms which in one instance had lasted thirteen, and in another seven or eight years. In one case there were severe intermittent attacks, as of acute cystitis. The administration of salicylate of soda, in doses of twenty to thirty grains twice or thrice daily, usually proved effectual; the urine became free from bacteria within a few days, and all the symptoms disappeared.

Sarcinæ.—Several observers have recognised the presence of sarcinæ in the urine, sometimes in sufficient numbers to form a greyish-white amorphous deposit. They resemble those derived from the stomach, except that they are smaller. They may occur in urine either acid or alkaline. The patient is generally troubled with symptoms of vesical or of renal disorder, but in some instances these have been probably attributable, not so much to the growth of the sarcinæ as to some concurrent disease, such as stricture or enlargement of the prostate. In a case recorded by Munk the sarcinæ were abundant in the summer, but almost wholly disappeared in the winter, notwithstanding that the patient, being paraplegic, was bedridden. Dr Bateman, of Norwich (cited by Dr Beale) met with a case in which the urine on four separate occasions contained sarcinæ for a few days at a time, apparently in connection with dyspepsia.

URINARY CALCULI AND GRAVEL

CALCULI—*Uric acid—Urate of soda—Oxalate of lime—Cystine—Xanthine—Indigo, &c.—Phosphate of lime—Carbonate of lime—Magnesian phosphate.* NEPHROLITHIASIS—*Lumbago—Hæmaturia—Solvent and operative treatment of calculi—Renal Colic—Obstruction—Atrophy—Hydronephrosis—Pyelitis and renal abscess.*

The materials of which calculi are constructed are, in the main, the same as those which constitute the various kinds of inorganic deposits from the urine, already described. We have now to consider how they undergo aggregation into calculi.

This question has been lately studied by Dr Vandyke Carter, Dr Beale, and Dr Ord. It has long been known that a stone is generally made up of concentric layers, which may differ among themselves in composition, and that the nucleus or central part may be quite distinct in character from the rest of its substance. Now, uric acid forms the chief ingredient of most calculi; according to Dr Roberts five out of six consist chiefly of this material. And until lately it was thought to be a frequent constituent of their nuclei. This, however, has now been shown to be a mistake. Even when the apparent nucleus really consists of uric acid, the microscope generally shows that the very centre of all has a different composition. It generally consists of an aggregation of spheroids or dumb-bells of oxalate of lime, or else of hedgehog-crystals of urate of soda. In countries where the bilharzia is endemic its ova frequently form a nucleus. In other cases it consists of inspissated mucus. or of a small blood-clot, or of casts of the renal tubes. Ebstein, in Ziemssen's 'Cyclopædia,' mentions a case in which he found in the urine a deposit of epithelial cells from the renal pelvis encrusted with uric acid, so as to give them a beautiful appearance, like scales from the wing of a butterfly; the patient, a woman, afterwards had symptoms of renal colic and passed concretions, but he does not state whether or not their nuclei were formed upon epithelial cells. Vesical calculi are sometimes moulded on foreign bodies introduced into the bladder from without. As regards the spheroids of oxalate of lime which form the nuclei of most calculi, Dr Beale maintains that they often have their origin within the tubes of the kidney. Not only has he found dumb-bells in the substance of casts, but he describes and figures microscopic calculi, already laminated, which he says he has many times seen in the renal tubes after death.

In the laminæ which generally make up the body of a calculus the materials are often laid down in a definite manner. Thus uric acid appears in the form of rods or columns, piled upon one another or arranged side by side; the urates are seen to form globules, themselves presenting concentric rings; oxalate of lime may form spheroids, and dumb-bells of this substance are sometimes embedded in laminæ consisting mainly of urates. Clear crystalline layers, however, make up a large part of some concretions. It does not seem to me, however, that these facts, which have been pointed out by Dr Vandyke Carter, indicate that calculi are anything else than "precipitates or aggregations of ordinary crystalline and amorphous deposits held together by mucus, &c.," though he thinks that such a theory is inadequate. When the principal ingredients of a stone are removed by solvents, a small quantity of organic matter is left; but to speak of this as an "organic basis" or "matrix" is, I think, to suggest misleading analogies. Dr Beale describes a remarkable instance in which a smooth, oval calculus, an inch and a half in length, was composed entirely of very small concretions, connected together by a whitish material; this, however, was altogether an exceptional specimen, for there is said to have been evidence of its presence in the urethra for fifty years before it was extracted by operation. In vol. xiii of the 'Pathological Society's Transactions' is a full account of this case by Mr Haynes Walton.

Few calculi consist entirely of one substance; and in many of them the main constituent of the several laminæ varies. Still conspicuous chemical distinctions exist between calculi; and there are corresponding differences in their physical characters.

1. *Uric acid calculi.*—These are hard bodies, of round or oval shape; smooth on the surface or finely tuberculated; of a yellowish, fawn, or reddish colour. Sometimes they are passed in enormous numbers while still very small, from the size of poppy seeds up to that of mustard seeds or split peas. Not infrequently several lithic acid stones, perhaps as large as marbles, are found in the pelvis of the kidney, or in the bladder. They then often have flat surfaces or facets, produced by contact with one another; the presence of such surfaces is always important as an indication that the concretion is not solitary. Calculi of lithic acid sometimes weigh three or four ounces.

2. *Urate of soda.*—These are soft concretions, which appear never to reach a large size, except by the addition of uric acid, or some other substance. Like crystalline deposits of urates they occur chiefly in children.

3. *Oxalate of lime.*—These calculi are characterised by their extreme hardness and by their rough irregular surface, whence the name of "mulberry calculi." They are, however, often passed per urethram while still small; they then appear as smooth, rounded, greyish or brown bodies which are compared to hemp seeds; or they may be covered with glistening crystals. As a cause of hæmaturia, pyelitis, or renal colic in middle-aged people, I should say, judging from my own experience, that such little calculi are far more frequent than those of any other kind. The larger "mulberry" stones are generally of a blackish-brown colour, irregularly rounded in form. Dr Roberts, however, says that stones of the same composition are sometimes oval, smooth, and of a bluish-grey colour. When crushed, they break into sharp, angular pieces. Mulberry calculi are usually solitary; I do not know that they are ever present in considerable numbers. Even of the small hemp-seed concretions it seldom happens that more than two of them are passed by the same patient and then usually at long intervals.

4. *Cystine.*—Calculi of this substance are usually egg-shaped; their surface is granular and glistens with minute crystals; they are of a honey-yellow colour, and on section look semi-translucent, like beeswax, and show indications of a radiating structure. It is curious that when exposed to daylight for a long time they often slowly acquire a delicate green hue. They are of rather soft consistence. They may reach a considerable size, weighing as much as three or four ounces, notwithstanding the low specific gravity of the material of which they are composed. Dr Roberts, however, describes two specimens which were passed *per urethram;* one was cylindrical and an inch and a quarter long, and weighed twenty-seven grains. They usually consist of pure cystine. But one mentioned by Dr Roberts had a nucleus of uric acid, and an outer layer made up of a mixture of uric acid and

cystine. And in the 'Path. Trans.' for 1880 (p. 182) there is described a cystine calculus which in all parts contained a minute proportion of oxalate of lime, and had in its interior a defined thin layer, consisting entirely of that salt: the nucleus was of cystine.

5. *Xanthine.*—This substance differs from hitherto mentioned constituents of calculi in not being certainly known as a urinary deposit. Dr Bence Jones alone, in 1862, found on two occasions what he believed to be xanthine as a sediment in the urine; the crystals were like those of uric acid, but they were at once dissolved when the fluid was warmed. Xanthine was first discovered by Dr Marcet about the year 1817, in a calculus. Altogether five instances of its occurrence in the form of concretions are on record. Its chemical composition is $C_5H_4N_4O_2$, *i.e.* uric acid, less one atom of oxygen. Consequently it was at one time termed "uric oxide." The characteristic test for it is analogous to the well-known murexide test for uric acid. When moistened with nitric acid it dissolves without effervescence, and on evaporation there is left a bright yellow residue; this, when cool, becomes violet red (not purple) if treated with a solution of caustic potass. It is insoluble in cold and almost insoluble in hot water, but is readily soluble in liquor ammoniæ or liquor potassæ.

Hitherto xanthine calculi seem never to have been found in the renal pelvis. In their physical characters they appear to resemble uric acid calculi very closely, except that they are of cinnamon colour.

6. *Indigo.*—This substance, again, can hardly be said to be known as a urinary deposit, though it sometimes colours lithates. As a concretion it has been found only once in the renal pelvis of a middle-aged woman, by Dr Ord ('Path. Trans.,' 1878). It formed a flat broad cake, like a lozenge in shape and size, weighing forty grains; its surface was partly dark brown, partly blue black; its section grey and polished. On paper it left a blue-black mark.

7. *Urostealith.*—This name was given by Haller ('Haller's Arch.,' Band ii) to certain soft elastic concretions, like india rubber, which were passed by a young man. Dr Moore, of Dublin, has since ('Dubl. Quar. Journ.,' xvii) met with similar specimens. And in the museum of the College of Surgeons there are two such which belonged to Hunter's collection. They were taken from the bladder, and the suggestion has been made that the fatty salts of lime of which they are entirely made up was formed by the decomposition of a solution of soap, which might perhaps have been injected for therapeutical purposes. This theory, however, seems not to be applicable to the other cases. In the 'Med.-Chir. Trans.' for 1872, Mr McCarthy has described certain calculi of peculiar form, eleven in number, which were taken from the left kidney of a woman after death. When first removed they felt soft and greasy, and they each consisted of a central globular body, with long tapering spines projecting from it. None of these appear to have been analyzed, but a somewhat similar concretion from the right kidney was found by Dr Tidy to contain 36·6 per cent. of fat and of cholesterine, the other chief ingredients being lithates (35 per cent.) and oxalates (9 per cent.). Similar specimens in the museum of the College of Surgeons are said to consist of oxalate of lime. Mr Benjamin Duke, of Clapham once showed me a number of similar calculi which he had taken from the body of a patient.

8. *Phosphate of lime or bone earth.*—All the seven varieties of calculi which I have been describing appear to occur in acid urine; certainly this is the case with such of them as are of great clinical importance. There is, therefore, a broad distinction between them and three varieties of which I have yet to speak, and which can only be formed when the urine is alkaline. One of these, which is, however, very rare, consists of phosphate of lime. Concretions formed of this substance are described by Dr Roberts as being

white and chalky in appearance, rather smooth on the surface with an earthy fracture. Their texture is sometimes loose, sometimes very compact. They vary in size from that of a pea to that of a hen's egg. In the museum of the Manchester Infirmary there is a laminated specimen in which bone earth alternates with uric acid. A peculiarity of calculi composed of phosphate of lime is, that while they require an alkaline urine for their production it yet must not be alkaline from ammonia, since, if it were, the triple phosphate could not fail to form, and to make up a large part of their substance. But even in urine which is alkaline from fixed alkali they seem to arise very seldom. Dr Roberts thinks that this is because bone earth, being uncrystalline, has by itself very little tendency to undergo agglomeration.

9. *Carbonate of lime.*—Concretions of this substance, again, are in the human subject very rarely seen. Dr Roberts says that when they do occur they are generally small very hard bodies, varying in size from the smallest visible up to that of a hazel nut; grey, yellowish, or bronze coloured; smooth on the surface, sometimes with a metallic lustre. Dr Haldane, of Edinburgh, once found a number of little calculi consisting of carbonate of lime in the dilated pelvis of the kidney of a man who had died from spinal abscess. Some years before Dr Roberts had met with a case in which immense numbers of precisely similar bodies were passed during life. The largest of them were of the size of mustard seeds, they were translucent and of an amber colour, and showed a laminated structure under the microscope. Dr Roberts was at first disposed to think that they came from the prostate, but Dr Haldane's case convinced him that they were of renal origin. The urine in which they were found was ammoniacal. In vol. xix of the 'Pathological Society's Transactions' a large renal calculus is described as composed of carbonate of lime, but in vol. xxviii it is stated that on a re-examination of the specimen it proved to consist of other materials.

10. *Mixed calcic and ammoniaco-magnesian phosphates* (fusible calculus).—In describing the alkaline fermentation of urine I have pointed out how, by the decomposition of urea into carbonate of ammonia it inevitably leads to the formation of the so-called triple phosphate of ammonia and magnesia, and how this salt and the amorphous phosphate of lime come down together as a precipitate, which has a strong tendency to agglomerate into a mortar-like mass. This substance sometimes concretes upon the surface of the inflamed vesical mucous membrane. It also accumulates upon any foreign body which is exposed to its action, and particularly upon calculi, of whatever nature, which at any period become washed by putrid urine. It seems comparatively seldom to form the actual starting-point of a stone, whether in the kidney or in the bladder. But, on the other hand, it often rapidly converts into a concretion of enormous size one that had before been of moderate dimensions. It is a soft friable substance; in the blowpipe flame it fuses into a kind of enamel, so that it is commonly spoken of as the "fusible calculus." In the bladder it may form stones weighing as much as twenty, thirty, or even forty ounces. In the kidney it is often moulded into the shape of the pelvis and calyces, having branches each with a funnel-shaped expansion at its extremity. Dr Gee has recorded (vol. xxxix, 'Med.-Chir. Trans.') a case in which such a concretion weighed thirty-six and a quarter ounces. It, or rather the remains of the kidney stretched over it, had been felt during life as an abdominal tumour of stony hardness. As is often the case, its surface was covered with brilliant crystals of pure triple phosphate, but these were of exceptional size and beauty, some of the prisms being half an inch long. The whole of the calculus was very hard and dense. It had a dark brown nucleus, which consisted mainly of oxalate of lime. Whether this also had a branched

form is not stated. But I believe that such "coral-like" masses, sending prolongations into the several renal calyces, are always composed of the mixed phosphates, and consequently are only found as a result of putrefaction of the urine.

Ætiology.—It will be apparent from this account of the different kinds of urinary calculi that in a patient whose urine is, and has been, acid, the only kinds that are at all frequently met with are those mainly composed either of uric acid, or of urates, or of oxalate of lime, or of alternating layers of two or of all of them. The ætiology of stone, therefore, resolves itself into the causes that favour the precipitation of these substances from the urine. It is true that for the formation of a stone there is required a nucleus, of which the nature may be altogether different from that of the rest of the mass. But, nevertheless, the conditions which cause calculous disease to be far more prevalent in some parts of the world than in others are believed to be chiefly such as lead to the deposition of uric acid or of its salts. The eastern counties of England, and especially Norfolk, are well known to yield a much larger proportion of cases of stone—at least of stone in the bladder—than any other districts of the country. Now, in his address on surgery, delivered before the British Medical Association, Mr Cadge, of Norwich, estimated ('Brit. Med. Journ.,' 1874, ii) that uric acid and the urates make up nine tenths of the calculi observed there, whereas for the whole of England a lower figure (usually five sixths) is generally given. Consequently, it seems not improbable that these ingredients may alone be concerned in producing the excessive number of cases observed in the eastern counties, the frequency of calculi formed of oxalate of lime, cystine, and other substances being perhaps no greater there than elsewhere. Mr Cadge could only call to mind three cases in which he had removed an oxalate of lime stone from an adult. In one of these cases only the outside of the stone consisted of that substance, the central part being uric acid; the patient had been recently residing in North Wales, having previously left Norfolk, probably with a uric acid stone already in his bladder. Another case occurred in a soldier who had only been for a short time in this country. The third was in a Norfolk man, but he also had resided elsewhere. As to why calculous disease should occur so frequently in the district in question, Mr Cadge could give no positive opinion. One point on which he insisted is that in young children it is almost entirely confined to the poor, being seldom or never seen in the more opulent classes of society. And he was inclined to think that a deficient supply of milk as food had much to do with it. The only young child of well-to-do parents whom he had treated for stone was said by the mother to have differed from all her other children in having never been able to take milk. He also believed that another important factor was the hardness of the drinking water. The hereditary influence sometimes seems to show itself in a very marked way. But persons who come to reside in Norfolk are said sometimes to acquire the tendency to calculus very rapidly, while others who leave the district lose it.

Stone is far more frequent in males than in females; but this applies especially to stone in the bladder, stone in the kidney being in Norfolk often seen in women. And so, as regards age, I am not aware that there are any trustworthy facts with regard to the frequency of renal calculus at different periods of life, though vesical calculi are well known to be much less common in adults than in children and in old people.

Nephrolithiasis.—The morbid conditions produced by the presence of gravel or calculi in the kidney are commonly classed together under the awkward name of nephrolithiasis. Strictly speaking, indeed, they may probably be said to depend in every case upon inflammation of the renal pelvis

But in practice the term *pyelitis* is usually reserved for those more severe cases in which suppuration occurs, and in which pus is either discharged with the urine, or collects so as to form an abscess in the lumbar region.

The following effects of gravel or calculi in the kidney require separate description:

1. *Pain in the loins,* or, as it is often called, *lumbago.*
2. *Hæmaturia.*
3. *Renal colic,* produced by the passage of a stone into the ureter.
4. *Obstructive anuria or suppression of urine,* caused by impaction of a calculus in one ureter, in cases in which the opposite kidney is from previous disease incapable of secreting urine.
5. *Unilateral atrophy of the kidney.*
6. *Hydronephrosis.*
7. *Pyuria* and *pyelitis,* with *pyonephrosis* and *perinephral abscess.*

It is, however, to be observed that there is scarcely one of these affections that may not sometimes be due to other causes, and not to nephrolithiasis. And, to save repetition, I shall in the present chapter discuss each of them fully, without attempting to limit my remarks to those cases in which calculi are present.

1. *Lumbago.*—In the mildest form of nephrolithiasis the principal symptom is a dull aching pain in the loins, such as is commonly called *lumbago.* Such a pain is, I believe, almost invariably the result of irritation of the kidneys by the urine, or by something deposited from it. Theoretically one might have expected that rheumatism affecting the lumbar muscles—like that which so commonly occurs in the shoulder-muscles—should be as frequent a cause of it. And one is often tempted to take this view by the patient's complaining that his back feels stiff, and that stooping greatly aggravates his discomfort or suffering. But, so far as I have seen, much more rapid relief is given in such cases by treatment directed to rendering the urine alkaline, than by any other kind of remedies. I have often seen the pain removed at once by a few full doses of the citrate of potass, repeated at short intervals. It may be a question whether the complaint is simply due to an over-acid state of the urine, or whether there is formed in the renal pelvis an actual sediment of uric acid, which is afterwards redissolved when the secretion becomes alkaline. One point in favour of the latter opinion is that the dull aching sensation is sometimes experienced only when the patient first wakes in the morning, ceasing directly when he gets into the upright posture. I know a patient who, if he ever takes a glass of beer, is almost sure to be reminded of it next day by an uncomfortable feeling in the loins.

2. *Hæmaturia.*—That urine contains blood may be obvious by its appearance; though, strictly speaking, one often cannot be sure that a case is not rather one of hæmoglobinuria until one has ascertained with the microscope that blood-discs are present. When the bleeding is very profuse the urine may look like pure blood. From this every gradation of colour may be observed down to the palest pink hue. But in many instances, when the colour is not in itself distinctive, there is a sediment the nature of which cannot be mistaken. On tilting the chamber-vessel from side to side, a granular-looking reddish-brown substance may be seen lying just within the edge of the fluid, and following its movements. And even when no such sediment is visible, the microscope may at once reveal the presence of blood in a drop of urine taken from the bottom of a vessel in which it has been standing for a little while, or indeed sometimes from the bulk of the urine directly after it is voided. It is impossible to insist too strongly upon the importance of microscopical examination in all cases in which hæmaturia may be suspected; without it the absence of blood should never be asserted.

Theoretically, albumen must be supposed to be always contained in urine in which there is blood. But the ordinary tests often fail to show it, when with the microscope blood-discs are at once seen. The only circumstances in which the results of a microscopical examination cannot be trusted are when the urine is of very low specific gravity or in a state of ammoniacal decomposition. Dr Roberts says that in such urines the blood-discs may be rapidly dissolved. In fluid having an acid reaction, and a good specific gravity they remain visible for several days.

Blood-discs in urine do not always retain their biconcave form. Sometimes they are shrivelled, with crenated edges, and sometimes they show protrusions, or perhaps give off masses of their protoplasm, which appear as microcytes. In dilute urine they are often connected with delicate globular bodies slightly larger than natural. Traube used to insist that the appearance of red discs altered into colourless rings rather smaller than their normal size was indicative of hæmorrhage from the substance of the kidneys, as contrasted with that from the urinary passages. The only things that can be mistaken for blood-discs in urine are compressed spirillæ, minute discoid forms of oxalate of lime, and (according to Dr Roberts) nuclei of renal epithelium. Dr Beale speaks of cases in which spirillæ looked so like blood-discs that great care was required to distinguish them.

In the exceptional cases in which the microscope fails to reveal the presence of blood, the spectroscope may be used (see p. 362) or chemical tests may be applied. One test is known as Heller's; it consists in rendering the urine alkaline by the addition of caustic potass or soda, and then boiling so as to precipitate the dissolved phosphates, which carry down with them any blood-colouring matter that the fluid may contain. Salkowski says that this reaction is very delicate, but that it also occurs with the colouring matter of rhubarb or senna. Another test which is commonly employed in this country is that of adding to a small quantity of urine a drop of freshly prepared tincture of guaiacum and shaking it up with a few drops of ozonic ether. If blood is present, a brilliant blue colour appears in the layer of ether that collects on the surface of the fluid when it has stood for a minute or so. The chief sources of fallacy lie in the fact that saliva, nasal mucus and iodide of potassium give the same reaction. According to Dr Mahomed ('Med.-Chir. Trans.,' lvii) this guaiacum test is even more delicate than that of the spectroscope.

It must not be forgotten that blood may be added to urine for purposes of deception. I have heard of such a case in which the microscope showed that the corpuscles which were present were the oval ones of a bird. I once checked an attempt of this kind, by remarking that it was necessary for me to see the urine passed; the patient, who had had a railway accident, subsequently confessed that he mixed with his urine blood that came from a cut upon the wrist.

In some cases hæmaturia is merely a symptom of a general disease such as purpura, scurvy, smallpox, or malarial fever. In some it is perhaps vicarious to menstruation or supplementary to the hæmorrhoidal flux, or to asthma; though for instances of such occurrences one has to go back to writers of the earlier part of the century. Rayer even cited examples of a periodical monthly hæmaturia in males. In many cases hæmaturia is only one among many symptoms of some disease of the bladder, or of the kidneys. I shall not here attempt to enter into the differential diagnosis of these several affections. But it must not be overlooked that from the presence of blood in the urinary passages in sufficient quantity to coagulate *in sitû* symptoms of various kinds may result, which simulate joint effects with the hæmorrhage of some antecedent disease. Thus a clot in the ureter may produce an attack of "renal colic" just like that caused by a calculus; clots in the bladder may give rise to dysuria, or even to retention of urine.

When coagula are passed in the urine they often clear up all doubt as to the seat of the hæmorrhage. Sometimes they are flat, having evidently been formed upon the floor of the bladder; sometimes cylindrical, when they come from a ureter. It is even stated by Bartels that they may retain the form of the renal calyces, though I must confess to a difficulty in understanding how they can have passed through the vesical orifice of the ureter without altering in shape; and in one instance that I can remember, in which they were supposed to have had such an origin, the bladder was afterwards found to be the source of the bleeding. Blood-casts of the uriniferous tubes of course prove that the hæmorrhage is derived from the renal cortex. Practically, the occurrence of bleeding from the substance of the kidney itself, in sufficient amount to require recognition as a form of hæmaturia, is limited to cases in which the organ has been lacerated by violence, to cases of poisoning by oil of turpentine or by cantharides, and to very rare cases in which quinine, as the result of idiosyncrasy in certain persons, produces a like effect, as has been observed by Dr Roberts (see also 'Brit. Med. Journ.,' Jan., 1870). A peculiar feature of the renal affection (generally described as an acute hyperæmia) which in some patients is set up by the application of a blister, is that fibrin is exuded in so large a quantity as to form transparent gelatinous clots in the bladder, which may obstruct the outflow of urine, so that catheterism is necessary to remove them. Two cases of this kind are related by Bartels.

Another circumstance that may aid in the diagnosis of the origin of hæmaturia is the way in which the blood escapes during micturition. Should bleeding occur into the urethra, the blood may precede the stream of urine, being washed out by it. On the other hand, when the bladder is the seat of disease, it may be especially towards the end of micturition that the urine is most deeply discoloured. Dr Beale speaks of cases in which persons, apparently healthy, day after day pass small quantities of blood, just as micturition is ceasing; it would seem that "the effort required to expel the last drop of urine causes the rupture of a few capillaries about the membranous part of the urethra or the neck of the bladder." In some instances this affection has been caused by undue sexual indulgence. It usually ceases after a time if the patient rests. When there is hæmorrhage from the renal pelvis the blood is always intimately mixed with the urine. But so it is likewise in many cases in which its origin is from the walls of the bladder.

If the urine, instead of being bright red, has a brownish hue, this shows that there has been time for the blood, after escaping from the vessels, to undergo chemical changes, and the inference is that it came from the renal pelvis if not from the kidney. But whenever the hæmorrhage is profuse the urine remains red until it is voided, and long afterwards.

From what has been stated it must be evident that in a large proportion of cases, including many of those in which the bleeding is most severe, there are no indications as to the seat of the disease, except such as consist in the presence of other symptoms, such as pain or dysuria. Now, in practice, almost the only vesical affections that give rise to profuse hæmaturia, as their sole symptom, are villous tumours and other forms of new growth, generally malignant. Such diseases occur chiefly in patients who have already reached middle age or passed beyond it. But in 1865, a child, only eighteen months old, died in Guy's Hospital, of the effects of a polypoid growth from the right side of the neck of the bladder. And in 1877 an autopsy was made in the case of a man, aged thirty-four, who said that ever since he was twelve years old he had suffered from hæmaturia, recurring at intervals of weeks or months, with greater or less severity. There was found to be a soft spindle-cell sarcoma, growing as a flat, slightly-lobulated tumour from the base of the bladder on one side. Villous cancers are the most common. It is a point of some importance that in a case of Murchison's ('Path. Trans.,' 1869), villous growths from the pelves of both kidneys were associated with a like affection of the bladder. Mr Davies-Colley once succeeded ('Clin. Soc. Trans.,' 1881) in removing through a perineal incision a villous growth which was attached to the left side of the posterior wall of the bladder, and which had a narrow pedicle two inches long. The patient was a man aged thirty-two, who had suffered for eight years from hæmaturia. He was completely cured by the operation. A point of interest in the case is that the patient sometimes passed blood at the beginning of micturition, sometimes at the end of it, the explanation no doubt being that the villi sometimes were nipped in the prostatic part of the urethra. Prof. Humphry has expressed the opinion that the occasional cessation of hæmaturia for a long period affords an indication in favour of the diagnosis of villous disease of the bladder as a cause of hæmaturia.

Renal hæmaturia—by which is understood hæmaturia dependent upon lesions of the mucous membranes of the renal pelvis—may be due to various causes, either calculi or malignant new growths, which may give rise to this symptom unaccompanied by any pain. When a patient passes blood in the urine without there being anything else to throw light upon the nature of the disease, the presumption is, I think, generally in favour of the presence of a renal calculus; but the possibility that cancer of the kidney may be developing itself must never be left out of consideration. It is, however, surprising how often one meets in practice with instances in which profuse hæmaturia, causing for the time the utmost alarm, occurs without there being any clue as to its source, but in which after two or three days it ceases, leaving the patient as well as ever. I can recall at least three cases of this kind which I have seen in consultation during the last few years, and in every one the cause remained to the last a mystery.

It scarcely ever happens that one has occasion to make an autopsy when hæmaturia has been the sole symptom produced by a renal calculus. But in 1881 an instance of this presented itself to me, and the case seems to be of great interest. A man, aged sixty-three, was admitted under my care with extreme wasting and cachexia, which led me to suspect the existence of malignant disease in some part of the body, although there were no symptoms to determine its locality. After a few days he was attacked with severe hæmaturia. This, however, quickly subsided, though afterwards pus appeared in the urine, which was also voided with undue frequency. Three weeks later he died. At the autopsy it was found that the cause of the wasting was cancer of the œsophagus. In one of the calyces of the left kidney a calculus was impacted. The lining of the renal pelvis was much thickened, œdematous, and of a deep purple colour from ecchymosis, looking like velvet. Probably a like condition is generally present when hæmaturia is the main symptom.

It is an interesting question whether bleeding ever occurs as a result of the irritation produced by mere granular deposits of uric acid, or of oxalate of lime, or whether the presence of larger concretion is necessary to give rise to the effect. A certain answer to this question can hardly be expected; but Sir Benjamin Brodie and others have taught that "red sand" (uric acid) is capable at times of causing hæmaturia. A point of considerable clinical importance is that jolting movements of the body are exceedingly apt to bring on, or to aggravate, hæmaturia in persons who have even small calculous concretions in the kidney. Not only does this occur after horse exercise, but even after riding in a carriage with springs. Even when no blood is obvious to the naked eye, it is always worth while to make a microscopic examination of the urine passed in such circumstances by a patient in whom renal calculus is suspected. In most cases of this kind there is lumbar pain, or pain referred to the front of the abdomen on one side, or to the groin. Another circumstance, to which Brodie drew special attention, is that the symptoms are sometimes referred mainly to the bladder. Micturition may be very frequent, and accompanied by a cutting pain in the neck of the bladder and in the urethra, so that fears are entertained of the presence of a vesical calculus.

In the *treatment* of hæmaturia, from whatever cause, rest in bed is of course essential. Cold should be applied as near the seat of the disease as possible; if it be from the kidney, ice poultices to the loin; if it be from the bladder, ice poultices to the hypogastric region of the abdomen, or injections of iced-water into the rectum or into the bladder itself. Prout found the injection of a solution of alum (twenty to forty grains in a pint of water) into the bladder very effectual when the hæmorrhage was vesical. As internal styptics, gallic acid, acetate of lead, ergot or alum may be employed. In some cases the tincture of perchloride of iron is particularly serviceable. Oil of turpentine, too, may succeed when all other drugs have failed. It would of course be wrong to give it should the blood come from the renal cortex, but in cases of hæmorrhage from the pelvis of the kidney there is no objection to its use. The hæmaturia caused by cantharides or by turpentine should be treated by cupping to the loins, warm poultices, diaphoretics, and purgatives.

In the treatment of recurrent or persistent hæmaturia attributed to renal calculus, the first thing is, if possible, to get rid of the cause of the disease. It has been thought that the entrance of a stone into the ureter, when it is not too large to pass through to the bladder, may be favoured by horse exercise, especially by hard trotting; but I should scarcely suppose that anyone would recommend this, since it could hardly fail to do harm in the event of the concretion being of any considerable size.

In all cases in which there is reason to hope that renal calculi consist of uric acid, or of urates, a fair trial should be given to the *solvent method*, elaborated by Dr Roberts. This observer made a careful series of experiments with calculi, outside the human body, exposing them to the action of a slow stream of a solution of carbonate of potass, which proved to be more effective than the carbonate of soda. With a liquid containing from forty to sixty grains of the alkali to the pint, he found that stones lost from 15 to 20 per cent. of their weight in twenty-four hours. Even with liquids containing twenty or thirty grains to the pint the solvent action was considerable. But what is very remarkable is that above the strength of sixty grains it ceased, in consequence of the formation of a white crust of alkaline biurate upon the surface of the concretion, which crust was often very tenacious and adherent, like white paint. The next step was to ascertain what doses of the vegetable salts of potass would give to the urine an alkalinity equivalent to about fifty grains of carbonate in the pint. And it was found that in adults this could be effected by the administration of forty to sixty grains of the acetate or citrate, dissolved in three or four ounces of water, every three hours; in children by about half the quantity. Some patients find that the acetate agrees with them better than the citrate, in others the reverse is the case. As the citrate of potass of the shops is apt to be impure, Dr Roberts advises that it should be prepared by neutralising a solution of the bicarbonate with crystallised citric acid; the formula given below* yields sixty grains of the citrate to the ounce.

It is not to be supposed that the urine passed by patients taking such doses of the potass salts can be maintained at an absolutely constant degree of alkalinity. On the contrary, it varies from hour to hour, but generally within the limits which correspond with the highest solvent action upon calculi. And Dr Roberts has found experimentally that such urine, when it is allowed to pass over a uric acid stone outside the body at blood-heat, dissolves it at the mean rate of twelve and a half grains in the twenty-four hours. Clinically, it is obvious that the best proof of the power of urine, when rendered alkaline in this manner, to act upon calculi within the body, is to be obtained in the case of vesical calculi, of which the presence and the approximate size can be determined by sounding before treatment is begun, and which can, if necessary, be removed by lithotomy afterwards. In one case Dr Roberts, after thirty-nine days' treatment, obtained the clearest evidence that a solvent action had been exerted; at the end of that time the cutting operation was performed and the stone was found to be eroded to a considerable extent, so that an incomplete layer of oxalate of lime was exposed, part of which was actually undermined. The proof of the efficacy of such treatment in the case of renal calculi is necessarily less complete. But there is obviously a presumption in its favour almost amounting to certainty. Dr Roberts relates the case of a gentleman who had repeatedly passed small brownish calculi, with symptoms of renal colic recurring with great regularity at intervals of three or four months. He came to Dr Roberts in July, 1860, with eleven such concretions which had come away a few days previously, which were found to consist of uric acid. His urine was made alkaline for a fortnight, and the result was that he passed no more concretions, nor had any return of the renal pain. During the following three months he took a drachm of citrate of potass in a tumbler of water each night and morning, and after this, a tumbler and a half of water without any salt in it at bedtime every night. Four years later he still remained entirely free from symptoms. Some years ago a similar case occurred to me. A man came to me with a number of little uric acid calculi which he had been passing frequently. I prescribed a vegetable salt of potass, and a fortnight later he brought me a single concretion, the only one he had passed, coated over with a white layer, which I thought at that time to be phosphatic, but which I now suppose to have consisted of the biurate, as described by Dr Roberts. Probably I had given too much of the remedy, but there seems to be little doubt that many other concretions must have been dissolved, for all the renal symptoms which had been troubling him disappeared, and he voided no more calculi while I saw him.

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* ℞ Potas. Bicarb. ʒxij; Acid. Citric. ʒviij, gr. xxiv; Aq. ad ʒxij.

The great drawback to this solvent treatment is that it is unfortunately altogether ineffectual when a stone consists of oxalate of lime, and that it fails even in the case of mixed calculi as soon as a complete layer of the oxalate is reached. It has been supposed that by rendering the urine alkaline one runs a risk of bringing about a deposition of phosphates, and so of actually augmenting the size of a calculus. But Dr Roberts has shown (see p. 349) that so long as the alkalinity is due to fixed alkali there is no danger of such a result. And as a matter of experience, he has found that after the treatment has been continuously carried out for three months, an oxalate of lime calculus in the bladder has remained entirely free from phosphatic incrustation. On the other hand, in the experiments already referred to, in which uric acid calculi were exposed outside the body to a slow stream of urine rendered alkaline by fixed alkali, and in which the calculi underwent solution, it was ascertained that as soon as ammoniacal decomposition of the urine occurred, a layer of mixed phosphates was deposited, and all further solvent action ceased. It is therefore useless to attempt Dr Roberts' plan of treatment unless the urine is acid. But even in cases in which putrefaction of the urine within the urinary passages has begun, the administration of the benzoate or of the salicylate of soda may perhaps sometimes succeed in arresting this change, and in restoring the natural acid state of the fluid, so as to bring the case again within the scope of solvent remedies.

In cases in which a renal calculus consists of oxalate of lime, medical treatment is, unfortunately, of very little avail. And it appears to me that among adults of middle age, the relative frequency of such calculi in proportion to that of uric acid concretions, is very much greater than would appear from the statements usually made in books (see p. 373). Perhaps these statements are based upon an enumeration of museum specimens, or upon statistics of cases of vesical calculi that have undergone surgical treatment. At any rate, in patients complaining of renal symptoms, I have met with many more examples of oxalate of lime calculi than of those of any other kind. When the presence of such a calculus is suspected, the only prospect of cure, apart from surgical operation, lies in the possibility that it may either pass down the ureter and be voided, or else become fixed in the renal pelvis or "encysted," so as to cause no further symptoms. The possibility of the latter occurrence was especially insisted on by Dr Rees in the Croonian Lectures for 1856. The tincture of iron is a medicine which may always be given with a prospect of advantage.

In all cases of protracted nephrolithiasis the question has to be entertained whether it may be possible to cure the disease by a surgical operation. The simplest plan is, of course, that of excising the stone through a lumbar incision. This has not infrequently been done in cases in which a perinephric abscess was present, or in which the dilated and suppurating pelvis of the kidney bulged freely in the loin. But cutting down upon a healthy kidney with the expectation of finding a calculus in it—a procedure which was condemned by Sir Benjamin Brodie as absurd and dangerous—has only very recently been proved to be feasible. The first case was one operated on by Mr Henry Morris at the Middlesex Hospital ('Clin. Trans.,' xiv) in 1880. The patient, a girl aged nineteen, was admitted under the care of Dr Coupland. She had for several years been liable to severe paroxysmal pain in the right lumbar region, which made her life as a domestic servant unendurable, and for at least two years her urine had often contained blood. Mr Morris cut down upon the kidney, and with his forefinger almost at once detected "something rounded, about the size of the uncut end of a pencil, causing a slight irregularity of the surface of the organ at a spot just a little behind the hilus." With a bistoury he incised the secreting substance of the kidney at this spot, and he then easily succeeded in removing a calculus by a slight scooping movement of the finger. There was no hæmorrhage of the least consequence. The concretion weighed thirty-one grains, it was triangular and flattened in shape, and studded with numerous small pointed elevations; it consisted of oxalate of lime. The patient made a good recovery. For more than two months urine flowed through the wound, the quantity varying from three to fourteen ounces in the twenty-four hours. A year later there was still a small sinus discharging a little pus. The girl was then in excellent health, and engaged in domestic service. In vol. xv of the 'Clinical Society's Transactions' two similar cases are recorded, each of which was no less successful than that of Mr Morris. One, by Mr Marcus Beck, is that of a young man, aged nineteen, who had suffered for twelve years from symptoms of stone in the left kidney, some pain in the loin increased by movement, hæmaturia, and great irritability of bladder. Mr Beck exposed the kidney, and on thrusting into the organ a darning-needle held in a pair of torsion-forceps, a stone was at once clearly felt. An incision was then made which was followed by an alarming jet of blood, but after pressure with a sponge for half a minute there was no further serious hæmorrhage, and the stone was extracted without difficulty after the incision had been enlarged by the introduction of a pair of polypus forceps and the separation of their blades. It consisted of alternating layers of uric acid and of phosphates. Rapid recovery took place, there being no escape of urine from the wound except for a brief period of four days. The other case, by Mr Butlin, possesses special interest from the fact that the symptoms were almost exclusively those of neuralgia of the right testicle which had continued for ten or twelve years. There was, indeed, some pain in the loin, and some tenderness in the corresponding side of the abdomen, but the urine never contained either pus or blood, though crystals of oxalate of lime were always present in it, and in many specimens a trace of albumen. The kidney having been exposed, a hard body was felt with the finger, which proved to be an oxalate of lime calculus. The wound quickly healed, but for some weeks after the operation the wound contained pus, apparently as the result of pyelitis. In volume xvi of the 'Clinical Society's Transactions' there is a fourth case by Mr May.

There can be no doubt that this operation, which is termed by Mr Morris *nephrolithotomy*, will in future be performed from time to time, as suitable cases present themselves. The chief difficulty is that of diagnosing with sufficient certainty that a stone is present, and that it is too large to pass down the ureter into the bladder. As a matter of fact, Mr Morris cites seven instances, in each of which an incision down to the kidney has been made without any stone being detected. And in one of these cases—that operated on by Mr Durham—the kidney was extirpated two years later, and it was then found that there really was no calculus. I well remember that patient; the terrible suffering which she underwent convinced all who saw her that there was some organic cause for it; yet on excision the kidney was found to be perfectly healthy, and at the autopsy no disease could be discovered. No doubt in future cases the discovery of the exact position of a calculus during the operation will be much facilitated by the suggestion made by Mr Barker, that after the kidney has been exposed a fine needle should be thrust through it at different points in the direction of the hilus, until the point is felt to impinge upon a hard substance.*

3. *Renal colic.*—The process by which a calculus passes down the ureter into the bladder is attended with special symptoms, which are conveniently designated by the name of renal colic. They often set in with extreme suddenness and violence, and constitute one of the most painful of all diseases. The patient is sometimes awakened by them from sleep; sometimes they are brought on by the jolting of a vehicle, or by some muscular effort, such as sneezing, coughing, running, jumping, or riding on horseback. The pain is generally described as running from one loin downwards in the direction of the ureter, but it may also spread over the whole of the abdomen, or radiate to the chest or to the shoulder-blade, or appear to run along the costal cartilages, or the iliac crest. Very commonly it extends down into the corresponding testicle, which becomes drawn up towards the inguinal canal, and is distinctly swollen as well as tender. There may be pain, too, along the inner side of the thigh, with numbness and tingling of the skin there. The suffering is often intense. The patient becomes faint and cold, and breaks out into a profuse sweat. The pulse becomes

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* [See cases of this operation and of nephrectomy by Prof. Czerny, Mr. Baker, Mr. Lucas, Mr. Barwell ('Internal. Cong.,' 1881, pp. 242—279), and since by Mr. Hayward (May, 1881), Thos. Jones, of Manchester (May, 1883), Howse (Jan., 1883), and Wm. Anderson ('Clin. Soc. Trans.,' 1884, p. 201). Also Sir Spencer Wells; 'Abdominal Tumours,' p. 199.—Ed.]

very rapid and small;* the breathing is quickened; the temperature may presently rise several degrees. Nausea and vomiting are sometimes marked symptoms; the matters rejected from the stomach becoming after a time bilious. I have heard Dr Rees say that he had seen jaundice occur. Epileptiform convulsions have sometimes been observed. In pregnant women abortion frequently takes place; cases are even recorded in which successive pregnancies have been brought to a termination by the supervention of attacks of renal colic at considerable intervals of time. In the vain hope of relieving the pain the patient adopts the most extraordinary positions. I remember a medical man, a friend of mine, telling me that the only attitude which seemed to give him any comfort was kneeling with the head bent over so as to touch the ground. But some persons prefer to lie on the side with the knees drawn up. Movements of the body generally increase the pain, and yet the restlessness is so great that it is often impossible for the patient to remain long in one position. The paroxysm, if it lasts long, is generally interrupted by remissions of the pain, which before long becomes as bad again as ever. Its duration is very variable; it may be over in the course of a few hours, or it may last for several days. Its termination is sometimes quite sudden; the patient, perhaps during a violent fit of retching, may experience a sensation as though he were stabbed, and from that moment the acute suffering may cease, the stone having slipped into the bladder.

Micturition during an attack of renal colic is generally frequent, and sometimes there is severe strangury, with burning pain in the urethra, or at the end of the meatus. Often only a few drops of urine are voided at a time, and they may be deeply coloured with blood. But if the kidney on the opposite side is healthy it may go on pouring out a normal secretion. Ebstein remarks that in cases of calculus pyelitis, in which the urine is habitually discoloured by blood and pus, the fact that it becomes normal when a stone is impacted in the ureter affords valuable evidence that the other kidney is not affected in the same way. But it often happens that calculi are present in both kidneys, so that the secretion from the one of which the ureter is still free is itself purulent, or blood-stained, or of low specific gravity and albuminous as the result of consecutive Bright's disease. Those cases in which at the time when an attack of renal colic occurs the opposite kidney is absent, or is atrophied so as to be unable to secrete any urine, will be specially described hereafter.

I am not sure whether the passage of a calculus is dangerous to life when the other kidney is healthy. Ebstein speaks of the possibility of its ulcerating through the walls of the ureter and escaping so as to set up a fatal peritonitis. But at any rate the case which he cites from Allan Webb ('Pathologia Indica,' 1846) was not uncomplicated, for "the vermiform appendix and the ureter were found ulcerated and adherent to one another and to the surrounding structures, and a large amount of pus had escaped from the ulcerated ureter into the abdominal cavity."

On the other hand, it does not seem that the subsidence of an attack of renal colic is necessarily a proof that the stone has passed into the bladder. Sometimes, perhaps, it undergoes disintegration in its course downwards, and the fragments of it escape in the urine without attracting notice. But in other cases it remains permanently impacted, and the kidney then undergoes atrophy or passes into a state of hydronephrosis, as will be presently described. The fact that the pain may cease while the apparent cause of it is still there, affords an argument in favour of an opinion held by Traube that it really is due, not so much to the direct irritation of the mucous membrane by the calculus, as to the peristaltic movements which take place in the dilated and over-distended ureter above. Dr Roberts, however, relates a case in which the track of a calculus could be followed along the whole length of the tube by the abrasions which it had produced. A ureter which has given passage to a stone may be found considerably wider than natural when death occurs from some altogether different cause, after the lapse perhaps of many years; I have seen the vesical orifice of the tube large enough to admit a thick probe. The occurrence of such dilatation of the tube explains the fact that when attacks of renal colic are frequently repeated, the later ones are comparatively slight and of much shorter duration. But of course a great deal depends upon the shape and the size of the calculus on each occasion. Sometimes the affection recurs at tolerably regular intervals. Sometimes a calculus is passed by a patient who never before showed any symptoms of urinary disorder. This is especially frequent when the concretion consists of oxalate of lime. An attack of renal colic sometimes puts an end to a pyelitis which had existed for a considerable time; in such a case one may infer that the renal pelvis contained a solitary stone which was the cause of the previous symptoms, and which has now escaped into the bladder. Prout, however, remarks that after the passage of an oxalate of lime calculus painful sensations often continue to be experienced for some time, so that he had found difficulty in convincing his patient that other calculi did not remain behind, when he was satisfied that there were none.

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* Traube, however, has recorded a case in which there were repeated attacks of renal colic, and in which the pulse was then always slow and full, and remarkably tense.

Again, it must not be supposed that the occurrence of an attack of renal colic is in itself proof of the passage of a stone into the ureter. In some cases (as, for example, in one recorded by Traube, in which the pain for some time returned every night, lasting about five hours, but in which no calculus was ever voided) it seems more likely that the attacks are due to irritation of the renal pelvis by a concretion, just as hepatic colic seems sometimes to arise without a gall-stone ever leaving the gall-bladder. But, again, precisely similar seizures may arise from the ureter becoming blocked in other ways; as, for instance, by echinococcus-vesicles, or even by clots of blood. Thus, in cases in which there is profuse hæmaturia, the occurrence of violent pain along the course of the ureter must not be taken as establishing the fact that the bleeding is due to a calculus rather than to cancer. Lastly, in a very interesting case recorded by Dr Dickinson in the 'Pathological Society's Transactions' for 1875, an abdominal aneurysm which had the ureter stretched over it gave rise for a long period to paroxysms of pain which were exactly like those of renal colic, and one of which was even attended with swelling of the testicle. In the immense majority of cases, however, renal colic is a symptom of stone.

The *treatment* of renal colic consists mainly in the administration of anodynes, though something may perhaps be done to facilitate the release of the calculus. Thus, if the abdomen is not too tumid, friction may be used in the course of the ureter; or, while the affected side is being rubbed, the patient may be held head downwards, as was done by Sir James Simpson with success in two cases, the concretion apparently falling back into the pelvis of the kidney. Venesection was formerly had recourse to, and numerous leeches were employed; but it is better to avoid such methods of relieving what is essentially a transitory affection. Large poultices applied to the loins sometimes help to remove the pain, but in other instances bags of ice have been more effectual. A hot bath sometimes seems to be of service. But our chief reliance must be placed in the free use of opium. The hypodermic injection of morphia is to be preferred on account of the rapidity and certainty of its action, but if this should be objected to, fifteen or twenty minims of tincture of opium may be thrown up into the rectum, or given by the mouth, unless there is vomiting. The dose will probably have to be repeated at rather frequent intervals. In some cases the inhalation of chloroform answers better than anything else.

4. *Obstructive anuria, or suppression of urine.*—In describing the renal affection of cholera (vol. i, p. 295) I had occasion to allude to the fact that the secretion of urine may, for a time, be completely suppressed, the patient voiding none, and none being found in the bladder when a catheter is passed. Poisoning by turpentine may produce a like effect; and in some remarkable instances it has been observed after an operation upon the urethra, or even after simple catheterism. I shall also have to speak of it as a symptom of suppurative nephritis, and of acute forms of Bright's disease. Such cases of "*non-obstructive suppression*" (as Dr Roberts terms them) end fatally in a few hours or in a day or two, unless the kidneys resume their function. When the affection is recovered from, the urine that is first passed is scanty and high coloured, and it generally contains albumen or even blood. The best treatment appears to consist in the use of the hot bath, or the application of hot mustard poultices to the loins, and the injection of hot gruel into the rectum.

In other cases, a temporary suppression of urine, without obstruction of the ureters, occurs as part of the general shock or collapse produced by the action of corrosive sublimate or of some other irritant poison, or by sudden lesions, such as perforation of the stomach, or rupture of the uterus. The renal affection is then relatively unimportant, passing off whenever the patient rallies, and generally leaving no ill-effects behind it.

According to Charcot, hysterical women are liable to a very different kind of suppression of urine, which he terms hysterical ischuria, and which may continue, almost without interruption, for a long time without seriously disturbing the health. He describes one of his patients as voiding less than a teaspoonful on an average each day for weeks together, whereas in the matters which she vomited urea was present.

It is, I think, difficult to believe that fraud was not practised in this and other like cases, although Charcot is convinced that he completely guarded himself against it.

A complete contrast to non-obstructive suppression of urine is afforded by what Dr Roberts terms "*obstructive suppression.*" When this occurs, the patient, instead of dying within a day or two, goes on for seven or eight days without any other grave symptoms, so that both he himself and his relations find it easy to imagine that there cannot be anything seriously the matter with him. He is calm and free from distress, with an unclouded intellect, and with natural pulse, respiration, and temperature. He may be able to take food fairly well, the tongue being clean and there being neither nausea nor vomiting. The muscular strength, however, may be observed to fail, and there is often marked sleeplessness at night. There is no desire to micturate, and sometimes no urine at all is voided. Generally, however, at very irregular intervals, the bladder discharges a few ounces, or even sometimes a pint or more of urine. This is always pale and watery and of very low specific gravity; and, unless blood is mixed with it, it is usually quite free from albumen. At the end of about a week symptoms appear, which are commonly followed by a fatal termination within two or three days at the latest. The most distinctive of these are muscular twitchings which Dr Roberts says are never wanting. Contraction of the pupils also constantly occurs. The muscular weakness now rapidly increases; and, as a result of its involving the respiratory muscles, the breathing is slow, panting, and laborious. The appetite is entirely lost, and the tongue and the palate become dry. There is increasing drowsiness, with short fitful snatches of sleep, and a little rambling delirium. Convulsions and coma rarely set in, the intellect being commonly preserved to the last, so that the patient has in more than one instance spoken sensibly the instant before his death. Diarrhœa is of quite exceptional occurrence; and so is severe vomiting. The skin is moist, and often sweats profusely; there is never any ammoniacal or urinous odour from the surface of the skin or with the breath; nor does the body give off such odours after death. In one instance slight general anasarca was observed when the suppression first took place, but it passed off entirely on the third day. The duration of life is stated by Dr Roberts to be, as a rule, from nine to eleven days, and he remarks that the passing of a few ounces, or even of two or three pints of a dilute urine does not seem to prolong it by more than a few hours. He knows of only three instances in which the patient survived beyond the eleventh day. In one of those cases, that of a man aged sixty-four, recorded by Rayer, death did not occur until the lapse of twenty-five days; another, that of a man aged seventy-three, recorded by Sir James Paget ('Clin. Soc. Trans.,' vol. ii) did not prove fatal for twenty-one days; the third, observed by Dr Roberts himself, in a woman, aged fifty-six, ended in death on the fifteenth day. The age of the patient does not appear to have any influence in accelerating or retarding the progress of the affection. Recovery has been known to occur in two or three cases in which there had been nearly complete suppression of urine for nine or ten days; in one of them the pupils had become contracted, or there was some mental confusion, but muscular twitchings had not made their appearance.

It is to Dr Roberts that we are indebted for the first complete account of the symptoms and causes of "obstructive suppression" of urine. But such cases had, of course, been observed before, although their characters had not been distinguished from those of non-obstructive suppression. It seems to me that the case recorded by Sir Henry Halford, and cited in 'Watson's Lectures,' must have belonged to this category, although it was much more rapid in its course, having apparently proved fatal in about three days. Sir Thomas Watson notes that patients affected with suppression of urine are chiefly persons who are advanced in life and inclined to corpulency.

The only instance that I know to have occurred at Guy's Hospital within the last thirty years is that of a man, aged forty-six, who in the year 1876 received a blow on the left side of the abdomen. This was followed by hæmaturia; two days later the urine became entirely suppressed and remained so until he died, seven days after the injury. In the course of the last twenty-four hours the muscles of his face were noticed to twitch, a profuse sweat broke out upon him, and he became unconscious.

In this case there was observed at the autopsy one unusual feature, namely, suppurative nephritis, and probably this accounts for its having reached a fatal termination more rapidly than those recorded by Dr Roberts. But the cause of the suppression of urine was found to be exactly that which he has insisted on as being almost always present, the obstruction of the ureter of one kidney by a calculus, when the other kidney is already incapable of secreting urine, some antecedent morbid condition. It is not, indeed, inconceivable that both ureters might simultaneously or in rapid succession become plugged with calculi, though I am not aware that such an occurrence has ever been verified by a post-mortem examination. The only other condition that can interfere with the flow of urine through both ureters at once (the two kidneys being healthy) and so cause an obstructive suppression of urine, is obliteration of their channels by pressure from without, as by cancer of the uterus or by some other disease of the pelvic organs, such as I shall have to discuss further on when I come to speak of hydronephrosis. Dr Roberts relates a few cases of this kind which ended fatally, and one in which after no urine had been secreted for seven days it flowed again naturally during the remaining four weeks of the patient's life. But I believe that in most cases due to such causes the renal cortex becomes atrophied or destroyed as the result of pressure or of a consecutive Bright's disease before complete obstruction of the ureters occurs, so that the symptoms and course of obstructive suppression seldom manifest themselves in a perfectly typical way.

In the regular form of obstructive suppression dependent upon blocking of the ureter of the only functionally active kidney possessed by the patient, it is to be noted that the renal pelvis does not become dilated to a considerable extent, and that the quantity of urine accumulated by it is by no means large. The substance of the kidney was in one of Dr Roberts's cases found to be much congested, but in another it was rather anæmic looking, though dotted on the surface with numerous blood-spots. I do not find any mention in human pathology of œdema of the kidney, and of a deeply ecchymosed state of the pelvis and of the upper part of the ureter, such as are described by Cohnheim as the usual consequences of ligature of the ureter in animals. The kidney, however, is generally of about twice the normal size, having undergone hypertrophy as the result of the overwork thrown upon it by the obsolescence of the opposite kidney during the months or years that may have passed since that organ became unable to take its share in the excretion of urine.

As I have already remarked above, whatever urine is formed by a kidney of which the ureter has become blocked, is pale, and of low specific gravity, and contains but a small percentage of urea. This is, perhaps, contrary to what one might have imagined to be the probable effect of such an occurrence, but it accords perfectly with the results of the experiments of Hermann upon animals. He showed that in dogs under a pressure of 2·4 inches of mercury the secretion of urine appears to cease entirely, and that when the pressure is removed the result is that a large quantity of watery urine is poured out, in which very little urea is present. Bartels relates the case of a young man who had suffered from previous attacks of renal colic, and who in one such attack had suppression of urine for five days. When this passed off, he voided in twenty-four hours more than 3000 cc., having a specific gravity of 1009, and containing numerous hyaline casts as well as albumen. Most observers seem now to think that the cessation of the activity of the kidney as soon as the pressure in the ureter and renal pelvis reaches a certain point is more apparent than real, the urine being really secreted, but being reabsorbed as fast as it is formed. I shall point out (p. 455) what an important bearing this view has upon the theory of uræmia, when taken in connection with Dr Roberts's observations as to the absence of the usual symptoms of uræmia in cases of obstructive suppression.

In the *treatment* of obstructive suppression, when it appears to be due to impaction of a calculus in one ureter, recourse should be had to those measures which we have seen to be sometimes effectual in aiding its expulsion downwards into the bladder, or its return upwards into the pelvis of the kidney, when there is renal colic. The abdomen in the course of the ureter may be well rubbed and kneaded, while the patient is in various positions,—standing, or lying, or inverted with his head downwards. But it is to be feared that the absence of pain in such cases means that the peristaltic movements of the ureter itself have ceased, and therefore that there is little chance of success from such means. In two of Dr Roberts's cases it is expressly noted that soon after the secretion of urine ceased the pain of which the patient had been complaining disappeared entirely. Consequently, it does not appear hopeful to employ hot baths, or chloroform inhalations, or anodynes of any kind, for the purpose of relaxing spasm.

There is, however, one method of treatment which seems never yet to have been attempted, but which I think is well deserving of trial. It is that of cutting down upon the kidney in the loin, and incising the ureter in the renal pelvis, so as to allow whatever fluid may be collected there to escape. The removal of pressure would probably at once be followed by an abundant secretion, and it is possible that a permanent fistulous opening in the loin might be created. Such an operation may not indeed be justifiable during the first few days after the suppression of urine has set in, on account of the possibility of spontaneous recovery; but there certainly can be no objection to it when at the end of a week muscular twitchings begin to appear. It is also a question whether it may not be practicable for the surgeon to remove an impacted calculus from the ureter. In the case that occurred at Guy's Hospital in 1876, the spot at which it was found on post-mortem examination was about one third of the distance down from the kidney. But in two cases recorded by Dr Roberts it lay just within the vesical orifice of the tube. Might it not be felt in such a position per rectum, and even be set free by a simple incision?

5. *Unilateral atrophy of the kidney.*—It is not at all uncommon in the post-mortem room to find one kidney shrunken to a mere thin flat relic, scarcely if at all bigger than the adjacent adrenal body, and weighing about an ounce or an ounce and a half. I have before me notes of twenty cases of this kind collected from our records at Guy's Hospital; and I have no doubt that, by a careful search, this number might be considerably increased. In several instances, the cause of death was some disease entirely unconnected with the urinary organs. The secretion of urine, in fact, takes place quite naturally under such circumstances, because the opposite kidney undergoes a compensatory enlargement, becoming as heavy as the two organs together normally should be. The nature of the process by which this enlargement is effected has been studied by different observers with discrepant results. Beumer, of Greifswald ('Virchow's Arch.,' 72) investigated it very carefully, and arrived at the conclusion that there is no demonstrable increase in size, whether in the glomeruli or in any of the tubes, so that it can only be ascribed to a new formation of the different structures of the organ. According to the strict terminology of Virchow, the compensatory change is a *hyperplasia* rather than a *hypertrophy.* I shall, however, continue to speak of it as hypertrophy, following the usual definition of that term.

However, the presence of an atrophied kidney is not without its effect, even when the kidney on the opposite side has thus become enlarged, so that it can secrete the full normal quantity of urine. For, if the ureter of this kidney should, from any cause, become obstructed, the necessary result, as we have already seen, is suppression of urine, instead of a mere attack of renal colic. And laceration of the region by violence is very likely to be followed by fatal results, as happened some years ago, in the case of a boy, admitted into the accident ward of Guy's Hospital, in which case during life it appeared a mystery why a unilateral injury should have such a serious effect. Moreover, it has appeared to me that the tissue of a kidney enlarged by compensating hypertrophy is unduly liable to become affected with Bright's disease; at any rate, in about one fourth of the twenty cases to which I have referred as presenting atrophy of one kidney there has been such disease of the opposite hypertrophied kidney. It would thus appear probable that the compensation is after all not perfect, and that the enlarged organ cannot for an indefinite length of time do the entire work of secreting urine without danger. The period of life at which the atrophy occurs might naturally be supposed to make a difference in the completeness of the hyperplasia; and, indeed, it is to be observed that Beumer's observations, already cited, were made in a case in which one kidney was congenitally absent, so that their applicability to cases of acquired atrophy may after all be disputed. But the liability of an enlarged solitary kidney to disease appears to be the same, whatever the cause of the enlargement. Among forty-eight instances of congenital absence of one kidney, collected by Beumer from different sources, there were no fewer than twenty in which the opposite kidney was found diseased. It most often was the seat of "chronic inflammation," but in many instances it contained calculi in the renal pelvis. I may remark incidentally that congenital absence of the kidney is probably much more rare than an acquired atrophy. It is accompanied by absence of the ureter, of the renal artery, and sometimes by other malformations.

A further interest attaches to acquired unilateral atrophy of the kidney in regard to its ætiology. In three of my twenty cases a calculus was found impacted in the corresponding ureter, and in two other cases calculi were present in the renal pelvis. In none of the remaining cases was any concretion found, nor was there any obstruction to the outflow of urine from the renal pelvis. Yet the pelvis and the calyces were dilated in no fewer than nine of them, and in two the ureter was dilated and thickened all the way down to the bladder. It seems difficult to avoid the inference that there had at some former period of the patient's life been a renal calculus, which either escaped through the natural passages or underwent disintegration, but which deranged the kidney sufficiently to cause it to waste. And this conclusion is greatly strengthened by the fact that in two other cases, in which it is not stated that any dilatation existed, there was a history of the performance of lithotomy many years previously; in one of them the operation had been done by Sir Astley Cooper when the patient was aged thirteen, forty-five years before his death. Lastly, in one instance in which neither the renal pelvis nor the ureter was enlarged, the vesical orifice of that tube was considerably lower than that on the opposite side, and lay nearer to the prostate, as though it had been forced downwards in the expulsion of a concretion. To me it seems probable that even in the remaining four cases in which the calyces and the pelvis appear not to have been dilated, the origin of the atrophy was the same. We shall presently find that this question has an important bearing upon the causation of hydronephrosis.

A very much more rare effect of the presence of a calculus in the renal pelvis is the replacement of the substance of the kidney by a mass of adipose tissue, having the shape of the healthy organ, and of about the same size as in a case described and figured by Dr Rickards, of Birmingham, in the 'Brit. Med. Journ.' for July 7, 1883.

6. *Hydronephrosis.*—We have seen that plugging of a ureter by a calculus, or obstruction of both ureters as the result of morbid processes of various kinds, does not necessarily lead to any considerable accumulation of fluid in those tubes, or in the renal pelvis. There are, however, cases in which such an accumulation occurs, and for these the name of *hydronephrosis,* originally suggested by Rayer, appears to be the most suitable, especially since it serves to distinguish them from cases of cystic disease of the kidneys, such as will be described afterwards.

Symptoms.—The slighter degrees of distension of the pelvis of the kidney appear to be unattended with symptoms. In the post-mortem room the earliest indication that the organ has been subjected to pressure from within is a diminution in the size of the pyramids, which, instead of nearly filling the calyces, become separated from them by broad intervals, and ultimately flattened, or even converted into hollows. With the microscope I once found that the tubes in the remains of the pyramids were bent into a regular series of wave-like curves. As this change in the pyramids goes on, the calyces and the renal pelvis begin, in their turn, to undergo dilatation. Sometimes the calyces stretch out of the hilus of the organ, so that the pelvis forms a sac situated nearer to the middle line of the body than the kidney itself, and sending finger-like processes into it; in a case that occurred at Guy's Hospital in 1876 such a sac lying beyond the kidney was found to hold a pint of fluid. Much more frequently the calyces and the pelvis, as they yield before the pressure of their contents, have the renal cortex expanded over them. Each calyx forms a somewhat egg-shaped cavity, communicating with the pelvis by a smooth orifice, and separated from the adjacent calyces by a tough fibrous membrane; the surface of the organ acquires a lobulated appearance, the lobules corresponding in number with these cavities; or, if the sac is very large, the septa between them may become perforated, and they may, perhaps, ultimately be broken down and form a single cavity.

In the meantime the secreting substance of the organ passes into the condition described as consecutive Bright's disease, or undergoes atrophy until at length no trace of it can be discovered, or at most only a few scattered relics here and there upon the walls of the sac. The ureter, too, may be dilated until it is as large as the finger of a glove, or even a coil of small intestine. In one or two instances it has actually been felt during life in an abdominal tumour.

The nature of the fluid contained in the sac of hydronephrosis varies in different cases. When the enlargement is but slight, as in most instances in which both kidneys are affected, it is still more or less dilute urine, which, however, may contain albumen, or be mixed with pus or blood. In those extreme cases which are generally unilateral, the fluid is sometimes pale and clear, sometimes stained with blood so as to resemble port wine. It is usually of lower specific gravity than normal urine, being in this respect like the fluid secreted in cases of "obstructive suppression." But in a remarkable case operated on by Czerny it must have had absolutely the same characters as the secretion of healthy kidneys, for the urine passed by the patient was in all respects natural, and yet extirpation of the hydronephrotic organ was followed by complete and fatal anuria, and on post-mortem examination it turned out that the opposite kidney had undergone atrophy and that its ureter was obliterated.

The solid matters dissolved in this fluid are generally urea, uric acid, and salts of the same composition as those that are found in urine. But in a case that came under my observation in 1876 neither urea nor uric acid could be detected in the fluid removed by tapping for a tumour believed to be hydronephrotic; and I believe that Sir Spencer Wells and Mr Cooper Rose ('Lancet,' 1868) have also met with instances in which urea has been absent. Mr Henry Morris ('Med.-Chir. Trans.,' 1876) cites cases in which the contents of hydronephrotic sacs in the fœtus have been devoid of urea. Albumen is commonly present in greater or less quantity. In some cases the fluid has been purulent, as in one described by Dr Pye-Smith ('Path. Soc. Trans.,' xxiii) in which six and a half pints of an opaque reddish fluid were drawn off by a trocar. The disease may then be called pyonephrosis, but I think that a special name is hardly needed for it. Dr Dickinson ('Path. Soc. Trans.,' xiii) has recorded a case in which a very large sac contained a gelatinous or colloid substance.

As I have already observed, the *causes* of hydronephrosis are often such as affect both kidneys simultaneously. Among these may be enumerated stricture of the urethra, enlargement of the prostate, various vesical affections (including villous disease of the bladder), pregnancy, prolapsus, or retroflexion of the uterus, and various kinds of pelvic tumour, especially cancer growing from the womb into the tissues around it, or involving the iliac glands. Cohnheim has recorded a remarkable case in a rachitic boy of eleven with contracted pelvis, in whom a double hydronephrosis was produced by the pressure of an enormously dilated rectum and sigmoid flexure. In such cases the renal affection is usually more marked on one side than the other. But there is almost always so much interference with the secreting action of the two kidneys that death occurs from such interference (if not from the primary disease) before the sac has become large enough to constitute an abdominal tumour capable of recognition during life. Mr Morris, however, relates a case of villous disease of the bladder in which a rounded swelling, of the size of the head of a small fœtus, was felt in the right loin. As a rule, the only clinical evidence of the renal affection is the pale watery condition of the urine, until perhaps convulsions or other uræmic symptoms set in and rapidly bring about a fatal termination. For example, in 1871, a woman, aged thirty-six, was lying in the uterine ward of Guy's Hospital with cancer, when she began to complain of severe headache; after two days she screamed out violently in the night and became unconscious; and in this state she remained until her death three days later. I made an autopsy, and found that the cause of her brain-symptoms was not cerebral hæmorrhage (as had been suspected) but uræmia; each kidney had its pelvis greatly dilated, its pyramids flattened, and its cortex pale, though not decidedly narrowed; probably the microscope would have revealed advanced morbid changes in it. In 1869 a woman, aged thirty-eight, was admitted into the clinical ward under my care, shivering violently and very cold, with a dry brown tongue and other typhoid symptoms, but with her mind clear, though her face had an excited look. She was said to have had prolapse of the uterus for a year, and her urine was found to contain pus. She died two days later, her temperature having been very low throughout. On post-mortem examination it was found that the womb had dragged down the vesical extremities of the ureters, and compressed them against the pubic arch. There was hydronephrosis on both sides, and the cortex of each kidney was greatly atrophied and had obviously lost its secreting structure to a great extent, being hard, pale, and smooth. A bilateral hydronephrosis may, however, also be produced by lesions affecting both ureters, at the same time or in succession; these will be discussed further on.

On the other hand, if the cause of the hypronephrosis is so situated as to affect the ureter leading from one kidney only, it may produce a tumour of very large size. The opposite kidney then undergoes hypertrophy, and as it may carry on the secretion of urine perfectly, there is nothing to prevent the development of the hydronephrosis to any conceivable extent.

Many cases are on record in which this affection has been mistaken for a large ovarian tumour, or even for ascites. The most remarkable of them all is perhaps one related by Mr Glass in the 'Philosophical Transactions' for 1747; the patient was a woman, aged twenty-three at the time of her death, who had been dropsical from birth; the abdomen then measured 6 feet 1 inch in circumference, and the sac contained thirty gallons of fluid. But in several other instances many pints have been taken from a hydronephrotic tumour during life, or have been found in it on post-mortem examination. Among the points which should distinguish such a tumour from an ovarian cyst are its having first made its appearance in the loin rather than near the pelvic brim, its having no pelvic connections, the presence of the colon in front of it. and the absence of resonant intestine in the loin. It has, in fact, all the characters of a renal tumour, as will be described when I come to speak of malignant disease of the kidney. Fluctuation is generally very well marked. Dr Roberts speaks of a soft undulating feel of the swelling in hydronephrosis, and of its outline as being sometimes distinctly lobulated. It may occupy a large part, or even the whole, of one side of the abdomen, extending across beyond the umbilicus downwards into the iliac fossa, and upwards beneath the costal cartilages.

In a patient under my care in 1883, there was a prominence in the epigastric and in the left hypochondriac region, while in the loin the bulging was but slight; and as there was obvious pulsation, with an audible bruit, I was at first disposed to think that the case was one of abdominal aneurysm. In that instance the history given by the patient himself contained one point which, if I had duly attended to it, would have saved me from all hesitation as to the nature of the disease. He said that on more than one occasion after the first appearance of the swelling it had for a time undergone a great diminution in size. He had not indeed noticed that under such circumstances there was any increased flow of urine, nor that the urine was altered in appearance. This spontaneous subsidence or disappearance of the tumour, when it is observed, constitutes by far the most important clinical character of hydronephrosis. In fact, if it is associated with an excessive discharge of fluid from the bladder, it may be said to be pathognomonic; and even without that corroborative evidence, the only cases in which a similar occurrence is likely to be met with are those in which an ovarian cyst ruptures into the uterus or into the intestine; but such events are probably always indicated by special signs, namely, the escape of fluid *per vaginam* on the one hand, or the entrance of air into the cyst on the other. Dr Roberts says that a sudden diminution in size or complete subsidence of the swelling was noted in nine out of twenty-five cases which he collected, in which the existence of a tumour was clinically ascertained. Sometimes the fluid discharged from the bladder has been observed to be of the appearance of "port wine." And in Dr Dickinson's case, a "nasty stuff" is said to have been passed in the urine, which would probably have been found to be gelatinous in character if attention had been given to it.

I have still to mention two other affections that may be mistaken for hydronephrosis, namely, hydatid of the kidney and renal cyst. Each of them is very rare, at least as giving rise to a palpable swelling. The former is almost certain to be set down to hydronephrosis, unless its nature is revealed by the escape of daughter-cysts *per urethram*, or by the characters of the fluid removed by paracentesis. The latter could probably be distinguished only after extirpation or on post-mortem examination; the two most striking instances that I know of are recorded by Mr Cæsar Hawkins ('Med.-Chir. Trans.,' xviii) and by Dr Hare ('Path. Soc. Trans.,' iv); in each of them the tumour filled the right side of the abdomen. Three or four other cases more or less similar are cited by Czerny in his list of cases of nephrectomy ('Trans. Internat. Congress,' 1881). Neither pain nor tenderness is necessarily present in hydronephrosis, though when the swelling is large it often causes a very distressing sensation of fulness or distension. In some cases pricking or shooting pains are complained of, which are perhaps due to local inflammatory changes in the peritoneum covering the sac. The colon is sometimes tightly stretched over the tumour, in such a way as to interfere with the free passage of its contents; thus in a case recorded by Dr Roberts the chief symptoms were at first those of intestinal obstruction, which recurred again and again during several years.

The lesions that may affect one ureter so as to cause a unilateral hydronephrosis, or each ureter so as to cause the same affection on both sides, vary in different cases. The most obvious of them is obstruction by a calculus. Thus in 1877 a man, aged forty-six, died in Guy's Hospital of dropsy due to Bright's disease affecting a hypertrophied left kidney; in the right ureter there was impacted a mulberry concretion an inch and three quarters in circumference; the right kidney was converted into a shining loculated cyst, with a smooth lining, upon which there was one little patch of renal substance about as large as a shilling still remaining. It is of course impossible that, after both ureters have been completely blocked by stones, the patient should live long enough to admit of the development of a double hydronephrosis. But in 1874 a boy, aged six, was in the hospital for stone, in the bladder, when he died of tonsillitis. Each ureter was greatly dilated and also the pelvis of each kidney. The right ureter was blocked by a second small calculus about an inch above its orifice; the left was free, so that the distension on that side had to be attributed either to interference with the downward flow of urine resulting from the vesical calculus, or else to the passage of that calculus at a time when the left ureter was free, or at least not entirely obstructed. In 1857 there died in the hospital a woman, aged fifty-six, who had a large fluctuating swelling in the left loin, and a smaller one in the right loin. Hydronephrosis was found to be present on both sides, and the pelvis of each kidney contained calculi, but it was only on the left side that impaction of a calculus in the ureter had taken place. This patient had been passing blood and pus in her urine all the while she was in the ward. But in most cases of hydronephrosis due to impaction of a calculus the urine is perfectly normal, though a history of former attacks of renal colic may perhaps be elicited, sometimes very far back. Rayer records the case of a man, aged sixty-four, who had for a long series of years enjoyed perfect health, but who at the age of twenty-two had had an illness attended with pain in the right kidney and along the ureter, and with hæmaturia.

Another cause of unilateral hydronephrosis is compression of the ureter, generally near the brim of the pelvis, by a thickened peritoneal band, the result of inflammation of the serous membrane. And sometimes the ureter is thickened and narrowed by changes in its own coats, the origin of which is no longer discoverable when the case comes to an autopsy, probably many years after their occurrence. A case of Dr Pye-Smith's ('Path. Trans.,' xxiii) to which I have already referred, and in which the ureter was found to be obliterated about an inch and a half below the pelvis of the kidney, appeared to be clearly traceable to a kick in the loins from a horse about two years previously; the injury had been followed at the time by hæmaturia. A similar instance, in which hydronephrosis in a boy, aged twelve, was directly traceable to a fall, has been recorded by Mr Croft ('Trans. Clin. Soc.,' xiv). In 1873 I made an autopsy in the case of a boy, aged four, who died with a calculus in his bladder; the ureter, which was as thick as a lead-pencil, was obliterated by an oblique cicatrix about an inch from its origin. There are also a good many instances in which no cause for the hydronephrosis can be made out, the ureter appearing perfectly free from obstruction in its whole course from the renal pelvis to the bladder. For my own part, I believe that in such instances there has generally, if not always, been at some former period a calculus, which has in the meantime undergone disintegration, or has been voided.

I have already drawn attention to the importance, with regard to this question, of the facts adduced at p. 391, in reference to the origin of atrophy of the kidney. I even think it doubtful whether some conditions that have been regarded by Dr Roberts and other observers as occasional causes of hydronephrosis are really capable of producing it, and whether calculi may not have been the true cause in the cases so interpreted. One such supposed cause is compression of the ureter by a supernumerary renal artery. Another is an obliquity of the origin of the ureter from the renal pelvis, causing a valve-like impediment. That such an appearance is not infrequently met with it is certain, and Dr Hare has recorded ('Med. Times and Gaz.,' 1857) a case in which the ureter on each side was coiled on itself—like a turn and a half of a corkscrew brought closely together—and adherent to the lower part of the sac. And there can be no doubt that this valvular arrangement of the ureter is the cause of the "intermitting" character of many hydronephrotic tumours, as well as of the fact that after puncture with a trocar the ureter sometimes becomes for a time again pervious. But what seems to me very doubtful is whether any congenital malformation is really present in cases of this kind, and whether the twisting of the tube is not rather a secondary result of the distension of it, just as one finds the duct of the gall-bladder distorted to an even greater extent and bound down by adhesions as the result of the passage of gall-stones through it. I have twice seen such a valvular condition of the upper orifice of the ureter when there was obstruction of the lower urinary passages; once in the case of an old man who died of the effects of stricture of the urethra, and in whom, although the ureter was not dilated, the pelvis of the left kidney formed a large pouch full of dark-brown fœtid fluid; and again in a fatal case of lithotomy, complicated with stricture.

A point upon which Cohnheim lays stress is the origin of the ureter from the side of the renal pelvis instead of from its lower end. The result of this, he says, is that so long as the patient is in an upright position the bladder receives only so much urine as overflows from the kidney. And he mentions the case of a woman so affected who for a long time passed scarcely any urine during the day, whereas she voided large quantities at night. This, however, surely proves too much, unless indeed the hydronephrosis was very considerable, and it must not be forgotten that in renal cirrhosis (to which the consecutive Bright's disease of hydronephrosis is very analogous) the nocturnal flow of urine is often very excessive. Cohnheim himself observes that before such cases come to an autopsy the conditions are so altered by the dilatation of the pelvis of the kidney as to render it impossible to say how the affection began. That hydronephrosis itself is sometimes congenital is well known: some cases in which the abdomen has been large from the time of birth, have been prolonged for years, although they far more often terminate fatally within the first few days or weeks. But, so far as I am aware, congenital hydronephrosis is always traceable to some definite malformation, such as closure of a ureter, or more rarely of the urethra. The cases in question do not, I think, lend any support to the view that obliquity or twisting of the upper end of the ureter, occurring as a malformation, can give rise to hydronephrosis. One point worthy of notice about the congenital form of the disease is that it is often associated with harelip, imperforate anus, club-foot, and other defects of development. The fact that closure of the outlets of the kidneys causes during intra-uterine life an accumulation of fluid seems to show that the secreting function of those organs must already be in a state of activity; and in a paper read before the Royal Medical and Chirurgical Society in 1876 ('Med.-Chir. Proc.,' vol. lix, p. 98), Mr Morris has argued for the view that they normally pour urine into the *liquor amnii*, whence it is absorbed into the blood of the mother, to be afterwards re-excreted by her urinary organs.

In a case that occurred at Guy's Hospital in 1868, Dr Moxon suggested a cause for hydronephrosis that, so far as I know, has not been observed by other pathologists. The left kidney was found by him after death to have the pyramids flattened, the pelvis and the calyces dilated. The patient was a man, aged twenty-two, who suffered from a lumbar abscess, and lay constantly on his left side with his pelvis raised upon an air-pillow, so that the tendency of fluid to gravitate within the ureter must have been from the bladder to the kidney, and not in the reverse direction.

Prognosis.—When hydronephrosis is bilateral, the patient is always in danger, since the structure as well as the functions of the secreting tissue of the two kidneys is inevitably interfered with; and in many cases the primary disease that has caused the obstruction to the escape of urine would in itself prove rapidly fatal, even without any such complication.

On the other hand, the course of unilateral hydronephrosis is commonly very chronic, and it scarcely ever in itself brings life to an end. In the case recorded by Mr Glass death was apparently due to pressure on the diaphragm and displacement of the thoracic viscera. In one observed by Mr Thompson, of Nottingham ('Path. Trans.,' xiii), it resulted from peritonitis set up by escape of the contents of the sac through an ulcerated aperture. In Dr Pye-Smith's case there had been communication with some part of the intestine, for the sac which had suppurated contained a mass of vegetable fibre, with bits of apple-core and part of a clove. In other fatal cases that have been recorded the cause of death has generally been either an independent disease (as, for example, acute tuberculosis in a case of Dr Hillier's) or else the supervention of some morbid process in the hypertrophied kidney on the opposite side of the body. Consequently, it is very unadvisable to interfere actively with hydronephrosis, unless the patient is unable to bear the pain and discomfort to which its presence may give rise.

Treatment.—In some few instances one can succeed in emptying the sac by rubbing the abdomen. Dr Roberts relates the case of a girl of eight, who came under his care with a soft fluctuating tumour in the left side, of about the size of a child's head. This was diligently manipulated in every direction, with the aid of a lubricating ointment, on alternate mornings. After the third time she suddenly passed abundant urine, the tumour forthwith subsided, and did not reappear while she remained under observation. A somewhat similar result was attained in a case recorded by Dr Broadbent ('Path. Soc. Trans.,' xvi) of double congenital hydronephrosis in an infant.

But when the sac is tense little can be hoped for from such a procedure; and there is often so much tenderness that it cannot be adopted. The only treatment then is to puncture the sac with a trocar. On the left side this may be done at a spot just anterior to the last intercostal space. But on the right side Mr Morris has shown ('Med.-Chir. Trans.,' lix) that there is danger of wounding the liver, and he advises that a point should be selected half way between the last rib and the crest of the ilium, and from two inches to two and a half inches behind the anterior superior spine. After the operation, fluid like that which has been withdrawn from the tumour sometimes passes for a time with the urine, showing that the ureter has again become pervious. But the sac almost always rapidly fills again, and may soon regain the same size as before. Thus in a case of double hydronephrosis, which was three times tapped by Fränkel, the patient did not micturate at all during from twelve to forty-eight hours after each tapping, the whole of the fluid secreted in the interval having doubtless accumulated in the two sacs. It is true that unilateral hydronephrosis is commonly attended with such extreme destruction of the renal cortex, that the organ can hardly be supposed still capable of forming urine. But experience seems to show that even in such cases fluid continues to be poured out by the walls of the sac. I think that the only case that I know of in which puncture, however often repeated, has led to permanent shrinking is that already referred to as having been recorded by Mr Croft ('Trans. Clin. Soc.,' xiv). In that instance, within fifty-four days of the accident which caused the disease, seventy-nine ounces of fluid had already collected. Paracentesis was performed eight times altogether, from three to four pints being removed each time. After the eighth operation, which occurred at three months' interval from the first, no further accumulation took place. In a case observed by Sir Spencer Wells ('Dubl. Quar. Jour.,'

1867) the patient, two months after a second tapping, passed two calculi *per urethram,* after which the tumour completely disappeared and did not return. On the other hand, there does not appear to be much fear of setting up suppuration in the sac by paracentesis, though this result has been known ('Path. Soc. Trans.,' xiii, Dr Little's case) to follow the attempt to cure the disease by making a fistulous opening. Czerny mentions ('Trans. Internat. Congress 1881') the case of a man in whom Gustav Simon had two years previously made such an opening, and who in 1881 was continuing to act as an attendant in the wards of the surgical clinique at Heidelberg.

Czerny, in his statistics of nephrectomy (loc. cit., p. 249), gives twelve cases in which that operation has been performed for hydronephrosis or for cyst of the kidney. Seven of them ended fatally, but this high mortality may perhaps be in part attributed to the fact that in five an erroneous diagnosis of ovarian tumour had been made. According to Mr Barker a lumbar incision is in cases of this kind preferable to one in the front of the abdomen. The case of Czerny referred to at p. 392, shows the importance of ascertaining that the opposite kidney retains its functional integrity. Probably this may be best done by making a preliminary opening into the sac, and allowing it to drain, so that after one can be certain that no fluid from it any longer descends the ureter, one can measure and test the urine passing into the bladder from the other organ.

7. *Pyuria and pyelitis.*—We have now to deal with those cases in which the presence of a stone in the kidney causes pus to appear in the urine, and in fact gives rise to a suppurative inflammation of the mucous membrane of the renal pelvis. But we must first digress so far as to consider the general subject of *pyuria* and briefly to indicate the various affections of the urinary organs that may lead to this symptom.

The presence of pus in urine commonly gives it a turbid opaque appearance, and on standing there is precipitated a dense whitish-yellow sediment which can only be mistaken for one of amorphous phosphate of lime or of the mixed amorphous urates, but may be distinguished from them by its microscopical characters, and also by remaining undissolved both after the addition of an acid, and also when the fluid is warmed. But in urine which has undergone the ammoniacal fermentation pus assumes a different character. It forms a viscid, tenacious substance, which glides out as a coherent mass when the fluid is poured from one vessel into another.

Formerly this was spoken of as "mucus," but it really always consists of altered pus. It often causes much pain and distress in passing through the urethra. One can artificially produce the same change, whenever urine contains any considerable quantity of pus, by adding *liquor potassæ* or *liquor ammoniæ* to the sediment in a test-tube, and shaking it. The pus at once loses its opaque yellow appearance, and becomes viscid. This in fact constitutes the one chemical test for pus, a test which in this country is commonly associated with the name of Dr Babington, though Leube attributes it to Donné. It is not applicable when the amount of deposit is small. In that case the microscope at once clears up any doubt as to its nature. It is an interesting fact that leucocytes in urine, even when it is alkaline and full of bacteria, sometimes retain for a considerable period their amœboid movements.

Whenever there is pus in urine, albumen is also present, being derived from the *liquor puris.* But if the quantity of pus is small, the albumen may not be discoverable by tests. It is often a very important practical question to determine whether the amount of albumen observed in urine containing pus is or is not greater than the pus itself accounts for; since, if it is greater, it affords evidence of the existence of Bright's disease in addition to the affection causing the pyuria. In various surgical affections of the urinary organs, the propriety of operative interference depends largely upon this point. Schwrikert has made with Leube some experiments throwing light upon it. Having added to urine 2 per cent. of pus, he found that in every microscopic field, prepared with fluid that had not been allowed to settle, there were from ten to fifteen leucocytes, and that the amount of albumen precipitated by boiling occupied about one tenth of the bulk of the urine. And his conclusion is that a coagulum of even one twentieth or one twenty-fifth is more than can be attributed to pus, unless at least some few pus-corpuscles are visible in each microscopic field. Tube-casts should also be carefully looked for.

Pyuria may be due to a great variety of affections. The possible presence of gonorrhœa must never be forgotten; nor, in females, that of leucorrhœa, which, however, is indicated by the large number of squamous epithelial cells, as well as of leucocytes, that are seen under the microscope. Cystitis, again, is a frequent cause, and one should remember that as the result of inflammation of the bladder there may sometimes be a good deal of pus in the urine without the patient complaining much of pain or having to micturate very frequently, especially if he has a stricture or an enlarged prostate.

Whatever may be the origin of cystitis, it is apt sooner or later to lead to an extension of inflammation upwards to one or both of the renal pelves. Thus, pyelitis is of frequent occurrence as part of the widespread change in the lining of the urinary organs that results from an ammoniacal decomposition of the urine within the body. It may also accompany various surgical diseases of the lower urinary passages. It may also be produced in association with nephritis by certain poisons, of which cantharides and turpentine are the chief. And slight forms of it are seen in connection with Bright's disease, and also (it is said) as the result of diabetes, or during the course of enteric fever and of other specific diseases. Again, it sometimes follows a blow or other injury to the loin. And, as Kaltenbach has particularly pointed out ('Arch. f. Gynäk.,' iii), it may develop itself after parturition, as the result, perhaps, of extension of inflammation from the pelvic organs. But none of these instances afford an example of the occurrence of a persistent and severe pyelitis independently of a like affection of other parts of the urinary tract of mucous membrane. And it appears to me very doubtful whether it is necessary, if tubercular cases (which will be described separately) be excluded, to admit any other cause except gravel or calculus for such forms of pyelitis as require special clinical recognition. It is true that one occasionally fails to discover any concretions in cases which have ended fatally after having been of long standing, or in which a surgical operation affords an opportunity of thoroughly exploring the diseased organ. But we have already found grounds for the belief that either unilateral atrophy of the kidney or hydronephrosis may result from calculi which subsequently disappear, and there seems to be no reason why the same should not also be true of pyelitis.

The symptoms that characterise "calculous pyelitis" are more or less severe, pain in the loin or in the abdomen, hæmaturia which generally recurs from time to time, and more or less constant pyuria. A case in point has been under my observation for some years. It is that of a gentleman who consulted me in 1876 for a slight pain or uneasy sensation in the left loin, for which I could find no cause. I told him, however, to watch his urine; and a short time afterwards he one day noticed some blood in it, and brought me the specimen to examine. I made his urine alkaline, and within two days the hæmaturia ceased, and has never returned. But from that time to the present the urine has almost constantly contained pus in small quantity, with apparently an excess of albumen; and crystals of oxalate of lime have almost always been discoverable in it, with sometimes crystals of lithic acid. In 1880 he passed a small oxalate of lime calculus, after which he was more free from pain than he had been for some years previously. At one time I thought I could detect a distinct enlargement of the left kidney, there being fulness, with a dull note on percussion, reaching as far forwards as a line drawn from the ninth costal cartilage to a little in front of the anterior superior spine of the ilium. Yet the general health has all along been good and the patient has all along been able to discharge responsible official duties. It very rarely happens that the disease assumes so mild a form as this. Generally speaking rigors recur from time to time; sometimes with regular quotidian periodicity. There is often considerable pyrexia, which may assume a hectic type. Diarrhœa may be persistent and intractable; or there may be obstinate constipation, from adhesion of the colon to the anterior surface of the affected kidney. When pyelitis runs on for a length of time the renal pelvis often becomes dilated into a large sac, which may be felt as an abdominal tumour, and may bulge into the loin as an elastic fluctuating mass, very painful and tender to the touch. If the ureter becomes from time to time blocked, this swelling may present great variations in size on different occasions, and there may be converse variations in the degree of pyuria, the urine being perhaps clear when the swelling is largest, whereas a subsidence of it may be accompanied by the escape of several ounces of pus into the bladder. In such cases of "pyonephrosis" the renal cortex probably always undergoes atrophy, or becomes shrunken by a process of consecutive Bright's disease. If there are calculi in both kidneys, as is often the case, this morbid process is of course quite sufficient to destroy life, with symptoms of uræmia. And even when the affection is limited to one side, the opposite kidney may, after undergoing hypertrophy, become affected with Bright's disease, either as the result of lardaceous changes in it, or independently of any such changes. But in other cases, after lasting a certain length of time, the inflammation subsides, and the kidney shrinks and dries up into a putty-like mass.

Again, many cases of pyelitis end fatally by the supervention of *perinephric abscess*. Inflammation probably never affects the renal pelvis for any considerable length of time, nor with any great degree of severity, without extending to the surrounding structures, which become indurated and matted together by new fibroid material. But in many instances, after a while the mucous membrane undergoes ulceration, and perforation with escape of urine and of pus takes place into the connective tissue. When this occurs there is usually a marked increase in the pyrexia and in the other general symptoms that may have previously resulted from the pyelitis. A fluctuating swelling may appear in the loins, with extreme local tenderness; and ultimately the skin may become reddened, and the abscess, if not opened by the surgeon, may point and break of its own accord. But in other cases the course taken by the suppuration is different. It may enter the sheath of the psoas muscle and make its way downwards into the groin, and even penetrate the hip-joint. A point on which Trousseau laid stress, is that when the psoas is affected the thigh is kept more or less rigidly flexed upon the pelvis; but in many instances it is impossible to determine during life the renal origin of cases of this kind. Or the pus may extend in front of the iliacus muscle, and point above Poupart's ligament. Or it may penetrate into the intestine; in such cases gas and fæcal matter often escape into the abscess-cavity, and subcutaneous emphysema may develop itself in the back, as was observed in two instances by Trousseau. Lastly, a perinephric abscess may burrow upwards through the diaphragm and the lung and discharge itself by the bronchial tubes. Conversely, an abscess starting from some other of the abdominal viscera may make its way into the urinary passages. The fact that pus is found in the urine must therefore not be taken as proof that the origin of a lumbar or of a psoas abscess is necessarily renal. Suppuration about the kidney appears sometimes to be the result of a fall or other injury to the loins, independently of any actual laceration of the organ.

When there is free discharge of pus in the loin, the inflammation sometimes gradually subsides, and recovery ensues. But as a rule, the prognosis of perinephric abscess is unfavourable, the patient becoming worn out by the drain of pus, by severe pain and hectic fever, or by lardaceous disease.

In the *treatment* of pyelitis, the first thing is, if possible, to remove its cause. When there is a chance that a uric acid calculus may be the starting-point of the inflammation, and when other conditions are favourable, a fair trial should be given to Dr Roberts's solvent treatment (see p. 381). Should there be reason to suspect the presence of a concretion of oxalate of line, however, this method is useless. But even then some amelioration of the symptoms may often be effected by the administration of the tincture of the perchloride of iron, or of such medicines as copaiba, or oil of sandal wood. In the case referred to on the last page, the free use of asparagus, when it could be obtained as an article of diet, appeared to be beneficial.

In all cases of protracted and severe pyelitis, the question of surgical interference has to be taken into serious consideration, and it should not be delayed too long; for the chances of recovery are much greater at an early period of the disease than when it is far advanced. If there is an abscess in the loin there can of course be no question as to the advisability of thoroughly exploring it, and of searching for and removing any calculi that may be present. And even when there is no evidence of anything more than pyonephrosis, it is almost always advisable to cut down upon the kidney in the loin, or to lay open the suppurating cavity, so as to allow of its thorough drainage. This operation is termed *nephrotomy*, and how successful it may sometimes be is well shown by a case related by Rosenberger, of Würzburg ('Trans. Internat. Congress 1881'). The patient, a medical man, had during the previous year had an incision made behind the anterior superior spine of the ilium, with discharge of several pints of offensive pus. He was reduced to a skeleton when the lumbar operation was performed. Yet, the cavity having been washed out with carbolic acid and a drainage-tube inserted, he gradually regained his healthy appearance and resumed his practice, a small sinus alone remaining. When, however, a large-branched calculus is found occupying the renal pelvis, there appears to be some doubt as to whether the attempt to extract it may not be as dangerous a procedure as the complete removal of the diseased organ. At any rate a case of this kind, which occurred to Mr Morrant Baker ('Trans. Internat. Congress' 1881), proved quickly fatal by shock and by hæmorrhage from the walls of the dilated renal pelvis. But nephrectomy itself is a most formidable operation in such circumstances. When there is a large pyonephric sac, a lumbar incision may fail to give room for its extirpation—as has been pointed out by Mr Howard Marsh ('Trans. Clin. Soc.,' 1882). Another difficulty is illustrated by two cases of Mr Barker's ('Med.-Chir. Trans.,' lxiv). In each of them the kidney was found to be surrounded by a mass of dense hard vascular tissue, which could not be removed, and which in one instance matted together the structures at the hilus in such a way that they could not be isolated. Lastly, there is the difficulty of making sure that the opposite kidney is capable of maintaining by itself a due secretion of urine. Cases have already been recorded in which fatal suppression of urine has occurred; for the organ affected with calculous pyelitis, of such severity as to justify its extirpation, was nevertheless the only functionally active kidney which the patient possessed. To obviate this risk Czerny has proposed to make two stages of the operation, first producing a urinary fistula, and after an interval proceeding to nephrectomy. Another suggestion made by Simon is that in female patients, after dilating the urethra, it may be possible to catheterise each ureter separately. And Teichmann, who for several years carried on elaborate investigations in the post-mortem room at Guy's Hospital, believes that even in the male subject he can, with an instrument introduced along the urethra, nip up the mouth of each ureter in turn, and so withdraw from the bladder the secretion of each kidney separately.

Suppurative Nephritis.—To complete the description of the effects of irritation of the kidneys, secondary to morbid changes in the urinary passages, one disease has still to be mentioned, namely, that in which abscesses form in the substance of the renal tissue. It is, indeed, probable that in most of the cases that have been commonly spoken of as examples of abscess of the kidney, the affection has really been pyonephrosis, complicated perhaps with suppuration in the perinephric structures. The occurrence of an abscess of any great size, limited to the tissue of the kidney itself, is obviously an impossibility; and I do not know of any evidence that large abscesses behind the organ, or around it ever arise by extension from a single starting-point of suppuration originally seated within its cortex, though this might seem probable. Perhaps there is no other condition in which a collection of pus really lying in the renal tissue attains so great a size as in some cases of ulcerative endocarditis, in which embolic infarctus soften and break down with more or less of sloughing.

The affection now to be discussed as suppurative nephritis is very different in its anatomical characters from those to which I have just been alluding. It consists in the presence of more or less numerous foci of inflammation within the kidney-substance, which appear upon the surface as minute round or irregular dots, and upon section of the organ as streaks, or lines, traversing the cortex to a greater or less depth, or even running continuously through its whole thickness as well as through the corresponding medulla. At an advanced stage there is well-formed creamy pus, which may escape as soon as the capsule is stripped off; when the disease is fatal at an early period, there is often only a soft milky-looking or pinkish-white material, which really consists of the secretory substance of the kidney infiltrated with leucocytes, but not yet completely destroyed. Surrounding the infiltrated or suppurating tracts there is much vascular injection, and all the renal tissue (if it was previously healthy) is commonly swollen and softened. Sometimes only one or two points of even commencing suppuration are discoverable in the whole of the organ; and the presence of this morbid condition is then not unlikely to be overlooked by a careless or unskilled pathologist. I dwell upon the fact, because I think that there is reason to believe that the recognition of even a single point of acute inflammation of the renal cortex may generally be taken as proof of the existence of a morbid change of sufficient severity to account for death. Some observers have indeed thought that the presence of scattered shrunken depressions in the renal cortex (which are not uncommonly found in cases in which suppurative nephritis is a very probable occurrence) affords proof that abscesses had actually been formed at a former period, and had subsided. But I am rather disposed to think that such apparent cicatrices are the results of a local chronic inflammatory process of an interstitial kind.

The *causes* of suppurative nephritis vary in different cases. Sometimes an affection having precisely the characters above described occurs as part of general pyæmia; and Dr Moxon says that he noticed that in cases of pyæmia resulting from perineal section or lithotomy, abscesses in the kidneys were more apt to occur than when pyæmia was due to lesions unconnected with the urinary organs. This observation is interesting, because some pathologists have been disposed to refer suppurative nephritis in general, even when obviously traceable to an inflammatory process spreading upwards from the bladder along the ureters, to an infection with some septic poison from without. Thus Dr Goodhart, in vol. xix of the 'Guy's Hospital Reports,' endeavoured to trace a connection between this affection and the presence of erysipelas in other cases in the same ward and at the same time. Moreover, as a matter of fact, I believe that the remarkable decline in the frequency of pyæmia at Guy's Hospital within the last few years has been accompanied by a corresponding decline in the frequency of suppurative nephritis. But in some cases, suppurative nephritis seems to occur as a primary morbid change, independently of any of the causes hitherto alluded to. I have heard Dr Moxon remark that he had more than once seen death immediately attributable to it in patients who had before been gradually sinking from some chronic disease. I remember that he cited these cases to me as possibly throwing light upon a case which I observed, and to which I shall allude, of suppurative nephritis occurring as a complication of diabetes. In Dr Goodhart's paper there are recorded three instances, in each of which, although some degree of inflammation of the bladder was found at the autopsy, it seems doubtful whether this was of sufficient intensity to account for so severe an affection of the kidneys; one was a case of enteric fever, another was a case of mitral disease unassociated during life with any urinary symptoms, the third was a case of severe superficial burns of the lower limbs. It is, however, not impossible that these cases, all of which occurred between the years 1864 and 1868, include those to which Dr Moxon was referring in his remarks above cited.

But in the vast majority of instances, suppurative nephritis is secondary to an affection of the urinary passages; either to some one of the common surgical diseases of the urethra or of the bladder, or else to paralysis of the bladder from some spinal lesion, or to compression of the ureter as the result of cancer or other disease of the uterus. As might be expected, most cases of this kind display in addition the mechanical effects of obstruction to the outflow of urine from the renal pelvis, on one or both sides, which have been fully described already. It is also to be noted that the suppurative nephritis itself is by no means always bilateral, and that even when it is so, its intensity is often very much greater on one side than on the other. In some instances the whole length of the urinary tract is obviously affected with inflammation, from the bladder to the mucous membrane covering the renal pyramids. But in others the lining of the ureter and of the renal pelvis is pale and appears altogether normal. Dr Dickinson is of opinion ('Med.-Chir. Trans.,' lvi) that the exciting cause of the nephritis is really the ammoniacal state of the urine resulting from its decomposition within the urinary passages. And, if correct, it is an important observation of his that suppurative nephritis as a secondary affection never occurs except in cases in which the urine has undergone this change. How rapidly the disease may develop itself is well shown by a case, which he narrates, of an old woman admitted into the hospital for a fracture of the femur, who two days later became unable to pass her water, so that a catheter had to be used. The urine drawn off was then natural, but very soon afterwards the urine became offensive, and death occurred within a week of the accident, three days after the urine had changed its character. Both kidneys were found to be suppurating. It would seem from these facts that the prevention of suppurative nephritis is included under prevention of ammoniacal fermentation of the urine (see p. 370).

The *symptoms* of suppurative nephritis are very obscure. Dr Dickinson describes them as follows:—"Shivering happens early and is apt to be repeated. Febrile symptoms rapidly follow with typhoid prostration. The pulse becomes rapidly feeble, the tongue dry and brown, the appetite absent. Vomiting is a frequent and often an urgent symptom. But hiccough seldom occurs, and sometimes diarrhœa or profuse sweating. The countenance becomes anxious and haggard, the complexion cadaverous or yellow, and (possibly with low delirium) the patient sinks into utter prostration, unconsciousness, and death." But I should doubt whether there is anything in these clinical features which would serve to distinguish a case of suppurative nephritis complicating severe cystitis and pyelitis from one of an equally intense inflammation of the bladder and of the pelves of the kidneys, with healthy renal cortex. What has struck me in the post-mortem room has rather been the insidious character of the affection under consideration, so that the patient has sometimes died quite unexpectedly, without having had any marked febrile or even cerebral symptoms. Thus I remember the case of a boy whom Mr Bryant was about to cut for stone in the theatre, when at the last moment the stone could not be felt, so that the operation was postponed. In the course of the following night he died, and at the autopsy I found both kidneys suppurating. In a case that occurred at Guy's Hospital in 1867 it is reported that the patient was "taciturn, and had a difficulty in making himself understood, but said he was better; he was out of bed, and a moment before his death he spoke sensibly to the nurse, saying 'good-bye.'"

The state of the urine throws but little light upon the *diagnosis* of suppurative nephritis. There may be a large quantity of pus in it, but one can never tell whether this is not rather the result of the pyelitis and of the cystitis which are so generally present at the same time. Indeed I should doubt whether the renal affection in itself ever causes a marked degree of pyuria. Dr Dickinson says that pain in the loin is sometimes present. In some cases the urinary secretion is scanty or even suppressed, but that is not conclusive evidence, since it might equally be produced by chronic renal cirrhosis.

In 1863 there occurred at Guy's Hospital a remarkable case of what appeared to be primary suppurative nephritis in a healthy subject. A young man, aged twenty, came in for suppression of urine; subsequently a little was secreted, but great dyspnœa set in and he died four days after his admission. The kidneys were found very large, weighing 19¼ oz., of a dark purple colour, and full of points of suppuration; one of them had upon its surface a small clot which suggested a doubt as to the possibility of some traumatic origin for the affection. The bladder was ecchymosed and contained a little opaque urine. The possibility of scarlet fever having been the cause of the renal affection was negatived. Dr Wilks, who made the autopsy, was obliged to leave the cause of the disease a mystery; in his report he expresses a regret that the spinal cord was not examined.

In 1859, a musician, aged thirty, was admitted for suppression of urine, which had set in five days previously, as the result of his being prevented from micturating for several hours while playing upon his instrument. At first he had passed a little urine, but the secretion had gradually ceased, and for four days after his admission he voided not a single drop, nor could any be obtained by a catheter. His breathing was hurried, and he became comatose. On the fourth day (the ninth from the beginning of his illness) he passed a large quantity of urine, but the next day he died.

At the autopsy it was found that the cortex of the left kidney was suppurating and at one spot there was actual sloughing; but the organ was also puckered by a chronic inflammatory process of older date, and adherent to its capsule. The right kidney was shrunken to the size of a walnut. The bladder was hypertrophied and slightly inflamed. The patient had given a history of a severe illness with urinary symptoms seven years before.

It might be thought that the determination of the amount of urea in the urine would probably throw light upon the state of the kidneys in many of the doubtful and obscure cases to which I have been alluding. This expectation, however, appears not to be verified by experience. Dr Goodhart ('Guy's Hosp. Rep.,' xix) records two instances in which he made quantitative analyses shortly before death, and found that the renal secretion contained thirteen or fourteen grains of urea to the ounce; a third patient passed in the twenty-four hours three pints of urine, with a total quantity of 592 grains of urea in it; and a fourth patient thirty ounces with 328 grains of urea in it. On the other hand, a man, who afterwards went out well, having had his bladder punctured *per rectum,* passed in the twenty-four hours likewise thirty ounces, but in this there were only 295 grains of urea, or less than ten grains per ounce.

DIABETES

Definition—Detection of glycose in the urine: the cupric, potash, and fermentation tests—The amount, specific gravity, and volumetric analysis of the urine—Its other characters—Symptoms of diabetes—Physiology of the formation of sugar in the body—Glycosuria—Theories of diabetes:—(1) *Escape of sugar*—(2) *Over-formation of sugar*—(3.) *Diminished destruction of sugar—Exalted glycogenetic function of the liver—Experimental glycosuria—General ætiology of diabetes—Complications—Modes of death—Prognosis—Treatment: by diet—by drugs.*

I have now to deal with a disease which until recently was regarded as an affection of the kidneys. We shall see that there are still great differences of opinion as to many points in the pathology of diabetes, and particularly as to the part of the liver in causing it. But no one will deny that the physiological basis from which all discussions as to the nature of the disease must start is the glycogenic function of the liver.

It will be convenient, however, before entering upon what must be a lengthy inquiry into the theory of diabetes, that I should first explain fully its symptoms and the mode of detecting it.

The most common clinical history of the disease is somewhat as follows: A man finds that his strength is failing him, he knows not why. He is the more surprised at this because his appetite is excellent, and, indeed, larger than it ever was before. Yet he loses flesh as well as muscular power. Then he notices that he passes an unusually large quantity of water, and evidently in connection with this that he is always thirsty and wanting to drink. He goes to a medical man, who makes a chemical examination of the urine, and finds that it contains a considerable quantity of a substance, *sugar,* which is absent from healthy urine, or present only in an exceedingly minute proportion. This justifies the diagnosis that the patient is suffering from Diabetes—or Diabetes Mellitus, as it is sometimes called by way of distinction from the disease termed Diabetes Insipidus, or Polyuria (*supra,* p. 344), in which the urine contains no sugar.

Tests for glycosuria.—There are several chemical processes by which it is possible to detect the presence of sugar in the urine, but the tests which are really used in practice are not very numerous. The chief among them is the *copper test,* which may be applied in several different ways. These are all based upon the fact that grape sugar or glucose—the form of sugar that is contained in the urine in diabetes—possesses the property of reducing the oxide of copper to a suboxide at the temperature of 212°. The oxide of copper is blue, and liquids containing it in solution have a deep blue colour. On the other hand, the suboxide is orange yellow, so that there is no difficulty in seeing whether reduction takes place or not. As I have just stated, there are various ways of employing the oxide of copper as a test for sugar. At Guy's Hospital we always use a solution which is a modification suggested by Dr Pavy of that known as Fehling's liquid. The way to prepare it is as follows:—640 grains of neutral tartrate of potass and 1280 grains of *potassa fusa* are dissolved in ten fluid ounces of distilled water, and 320 grains of sulphate of copper are dissolved in other ten fluid ounces of water. The solution of sulphate of copper is then poured into that of the potass salt; the result is the formation of a clear liquid of a deep blue colour.

The way to use this test is a follows:—About a drachm of the liquid is placed in a test-tube, and heated until it begins to boil. A drop or two of the urine is then added, and if no change is observed, a further quantity of urine, until this equals that of the copper solution. The test-tube is then again heated until the liquid in it reaches the boiling point. After this it is allowed to cool, there being no advantage, but rather the contrary, in continuing to boil it. If any sugar were contained in the urine, the liquid, before it cools, will be found to have deposited a yellow or red sediment of the suboxide of copper.

There are several points which require comment in the rules for the application of the copper test. In the first place, one reason for applying heat to the copper solution first, and not to the urine, is that when the solution has been kept for some time exposed to light it is sometimes found to undergo a slight reduction when boiled by itself.

According to Dr Pavy the addition of a fragment of caustic potass to the liquid, when it has become deteriorated by keeping, will render it again as fit for use as ever. Another reason for heating the liquid first, is that healthy urine, if it happens to contain a large proportion of solids, possesses when boiling the property of decolorising the copper solution if added to it drop by drop. The mixed liquids have then an amber yellow colour, and may often contain flocculi of phosphates (precipitated by the alkali of the test). I have repeatedly found that clinical clerks and others regard this change as proof of the presence of sugar; but there could be no greater mistake. Indeed Dr William Roberts, of Manchester, says that when the blue colour of the copper solution has been discharged by the other ingredients of the urine, it is no longer possible for any sugar that may be present to precipitate the suboxide. This peculiar change would doubtless commonly render useless the careless application of the test to diabetic urine, were it not for the fact that such urine generally contains so small a proportion of the urinary solids as to be unable to decolorise the copper solution.

The reasons for adding urine in the exact way prescribed are thus given by Dr Roberts, who has devoted much pains to the elaboration of the details of the process. In the production of a deposit of the suboxide it is necessary that the sugar should not be in excess. Unaltered sugar has the property of dissolving the suboxide of copper. Hence, if urine containing a large proportion of sugar be added to the copper solution in considerable quantity no precipitate will result, but only an opaque yellow solution. But if only a drop or two of the saccharine urine is added, a deposit is produced, which for some reason is then always of a red colour.

On the other hand, when the urine contains only a small proportion of sugar, it must be added in larger quantity. As soon as the boiling point is reached, the liquid then changes to an intense opaque yellowish green, and a bright yellow deposit is slowly formed. Dr Roberts has determined the exact limits of the application of this test. He finds that one tenth of a grain per fluid ounce can with certainty be detected by it. According to Dr Pavy, the yellow precipitate sometimes fails to be produced when there is albumen in the urine; the latter must then be boiled and filtered before it is added to the copper solution. It is well known that many other organic substances besides sugar are capable of reducing the oxide of copper; but none of these are ever present in the urine, with the exception of leucine, uric acid, and chloroform. And of these uric acid alone could be supposed to be likely to occasion a fallacy in the detection of diabetes. It is certain, however, that urine containing an excess of uric acid does not generally give rise to a change in the copper solution at all resembling that which would be produced by sugar; but Dr Pavy states that he has found it to occasion a slight deposit of the suboxide.

The copper test for sugar fulfils all practical requirements, so far as accuracy and delicacy are concerned. Still there is an advantage in one's having another method of detecting diabetes, upon which one can fall back for corroboration in any doubtful case. And that which is most commonly used for this purpose is known as *Moore's test.* It consists in boiling one or two drachms of the urine in a test-tube with half its bulk of liquor potassæ. The ebullition must be kept up for some little time, and as it goes on the liquid becomes darker, passing through a series of colours which are almost exactly those of different kinds of sherry wine, until it at last becomes brown or even black. Now this test is not a very delicate one; according to Dr Roberts it does not succeed with urine containing a quantity of sugar less than a grain and a half in the ounce. Moreover, urine containing albumen, and generally all high-coloured urines, become somewhat darker when boiled with liquor potassæ, even though they contain no sugar. And if the potass solution has lead in it, as is often the case, albuminous urine sometimes gives with it a dark porter-brown colour, which has been mistaken for that which sugar would have produced. Moore's test is therefore valuable chiefly as a preliminary test. But when it yields a negative result, this may generally be depended upon.

Another test for sugar which may be used for corroboration is the *fermentation test.* A small quantity of yeast, which must be first thoroughly washed so that any adhering starch or sugar may be removed, is added to the urine, and this is set aside in a warm place, with a controul glass. When sugar is contained in it this is presently decomposed into alcohol and carbonic acid. The latter if found in any quantity, is given off as a gas, and may be readily collected. For this purpose, all that is needed is that the urine should be made to fill a test-tube, and that this should be then inverted in a saucer, and kept in position by a clamp. In a few hours it will be found that the liquid has receded from the upper or closed end of the tube, in consequence of the accumulation of the carbonic acid gas. (See also Picric Acid test, p. 434.)

Amount of urine.—By the employment of two of these tests, or even of one of them, it is always easy to determine whether a patient is or is not suffering from diabetes; but this is by no means sufficient. Both for prognosis and treatment it is absolutely necessary that an estimate should be procured, not only of the proportionate amount of sugar which the urine contains, but also of the total amount of sugar which is excreted by the kidneys in the twenty-four hours. With this object in view all the urine which the patient passes must be carefully collected and measured every day for some days in succession. Now, the proportionate amount of sugar contained in the urine is by no means uniform at different periods of the twenty-four hours. All that is passed each day must therefore be collected in a single vessel, and from this a sample must be taken for accurate analysis. I have already stated that in diabetes the quantity of urine is much larger than in health. Instead of being three pints in the twenty-four hours, or less, it is often as much as fifteen pints; and according to Sir Thomas Watson, cases are recorded in which seventy pints have been passed. Dr Pavy has himself seen a case in which thirty-two pints were collected and measured in one day.

Specific gravity.—The step next taken is generally that of determining the specific gravity of the urine. This is very easily done by methods which I will describe in discussing the diseases of the kidneys. It will be readily understood that in diabetes the specific gravity of the urine is higher than normal. Instead of being between 1015 and 1025, it is from 1030 to 1040 or 1045. Some writers have said that it may reach 1060 or 1070. But, according to Dr Pavy, the maximum is a little above 1050. From the specific gravity of the urine in a case of diabetes one can form a rough estimate of the proportionate quantity of sugar contained in it; but the relations between them are by no means absolutely constant.

Quantitative analysis.—There are two or three different methods by which the amount of sugar present in a certain quantity of urine can be accurately determined. That which I always myself employ was introduced by Dr Pavy. It consists in ascertaining how many minims of urine are required to reduce the whole of the oxide of copper in 100 minims of the copper solution, the formula for which I gave a few pages back. The copper solution is first measured by a pipette into a porcelain capsule. Into it is then dropped a fragment of caustic potass, of about twice the size of a pea, this having the effect of causing the reduced oxide afterwards to fall in a dense form, so that the colour of the remaining liquid can be more readily observed. The capsule is next heated by a spirit lamp until it boils steadily, a pipette graduated to hold 100 minims, with subdivisions, is in the meanwhile charged with the urine and this is now allowed to flow drop by drop into the boiling copper solution, which is kept constantly stirred with a glass rod. If sugar be present the yellow or red oxide of copper gradually appears in greater quantity, but as soon as it is formed it settles, leaving the liquid still blue. At length, however, the blue colour is entirely removed, being replaced by an orange or an orange-red. At the moment when this occurs the operation is suspended, and a glance at the pipette shows how much urine has been used. The copper solution is of such a strength that exactly half a grain of sugar is required to decolourize 100 minims of it. Thus, there is half a grain of sugar in the quantity of urine that has been dropped from the pipette. It is a matter of the simplest calculation to determine the amount of sugar that must be contained in each ounce of urine or in the whole amount excreted daily. In his book on diabetes Dr Pavy gives a table by which the trouble of making this calculation may be saved. This process is constantly employed at Guy's Hospital, where the clinical clerks habitually carry it out. It takes only a very short time, and after a few trials anyone can learn to do it with a sufficient amount of accuracy. If the urine be at all highly charged with sugar it is advisable to dilute it with from two to four parts of water before employing it for analysis, of course, making the necessary correction afterwards.*

Most writers recommend a method of determining the quantity of sugar in urine which is essentially the same as that which I have just described. But Dr Roberts has shown that another very easy plan is to ferment the urine with a little yeast, and next day to take its specific gravity and to compare it with that of the same urine in its unfermented state. For each grain of sugar per fluid ounce one degree of density is lost by the process of fermentation. Dr Roberts says that this method yields very fairly accurate results, and its performance requires no technical skill, the only objection to it is the delay, the result being obtained only after the lapse of twenty-four hours.

The amount of sugar contained in the urine in diabetes varies from the smallest trace up to forty-eight grains in the ounce. Dr Pavy believes that this proportion is never exceeded, and that when it has been reached a further increase in the quantity of sugar requiring to be excreted by the kidneys leads necessarily to an augmented flow of urine.

The total quantity of sugar excreted daily of course shows corresponding difference. Dr Dickinson speaks of cases in which it amounted to fifty ounces.

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* **[Dr Pavy has since devised an ammoniated cupric solution which we have found very satisfactory in the wards. See 'Lancet,' June, 1884.—Ed.]**

Some other characters of the urine in diabetes remain to be mentioned. It is generally pale, and the more so the greater the quantity that is passed. When this is very large the urine may look just like spring water. Again, it is almost always clear, depositing no urates even when it has cooled. This fact has a practical value because it sometimes enables us to form an opinion as to when the disease began. Dr Prout used to ask his patients how long a time had passed since the urine became thick on cooling, and if such turbidity of the urine had previously been frequently observed he would date the commencement of the diabetes from the time when it ceased to occur. On the other hand, when the amount of sugar is great it may itself crystallize as the urine dries. Sir Thomas Watson mentions the case of a woman who was alarmed at finding her black worsted stockings sticky and covered with a white dust from this cause.

Urine containing sugar has a sweet taste, a fact which was first pointed out by Dr Willis about the year 1674, when of course the methods of detecting sugar which are now practised were not known. At the present day this point is not often investigated, but it is said that flies and wasps are attracted to vessels containing diabetic urine. The secretion has a peculiar odour, which was compared by Dr Prout to that of sweet hay or milk, by Sir Thomas Watson to the faint smell of certain apples, or of an apple chamber. The *Torula cerevisiæ,* or yeast plant, forms in diabetic urine when it is left freely exposed to the air in a warm place; and the sporules of this fungus may be readily detected with the microscope. Formerly its development was supposed to be a proof that a specimen of urine contained sugar, but even in healthy urine sporules may be found, which are, indeed, said to belong to the *Penicillium glaucum,* but which are undistinguishable from those of the torula.

When a patient suffering from diabetes is attacked by any intercurrent febrile disease, the urine often, but not always, becomes for the time free from sugar. This is a point of considerable theoretical interest, as tending to confirm the view that the disease depends upon a perversion of the glycogenic function of the liver; for it has been shown that when fever arises in healthy subjects, glycogen disappears from the liver.

Symptoms.—The chief symptoms of diabetes have already been enumerated at p. 407. Foremost amongst these must be placed *muscular weakness.* This is often extreme. It is by no means to be regarded as a mere result of the wasting of the muscles which generally accompanies the disease. In a series of experiments which Dr Pavy made with various kinds of diet on a man affected with diabetes, he found that as soon as the patient was put upon food which caused him to pass an excessive quantity of sugar, he complained that he had no life or energy in him. Another patient when admitted was so weak that he could not stand alone; in about three weeks time under treatment he had gained strength to such an extent that he ran to the end of the ward and back to show what he could do. A loss of virility is another frequent effect of the disease, and in women suppression of the catamenia. Natural vigour of character is also replaced by feebleness, moral and intellectual.

Again *thirst,* is one of the earliest and most persistent symptoms of diabetes. The patient generally drinks from eight to twelve pints a day, but sometimes as much as twenty-five or even thirty-five pints. Yet even this does not satisfy the craving. The mouth and fauces are also the seat of a sensation of dryness, which causes great discomfort. Dr Pavy says that the way in which the patient keeps rolling the tongue about in the mouth, and the sound which it produces by sticking to the palate from time to time, may be recognised as signs of the disease. The tongue itself is often intensely red and clean, and sometimes it is fissured. Occasionally a subjective sensation is experienced of a sweet taste in the mouth.

An increased *appetite* for solid food is by no means so constant a symptom as thirst. Sometimes the appetite is enormous, but in the later stages of diabetes there may be a loathing of all kinds of food. The teeth generally become carious, and the gums swollen, loose, and inclined to bleed. Very often the patient complains of a peculiar and distressing sensation of hollowness at the pit of the stomach. Sometimes vomiting and symptoms of dyspepsia are present, but, as a rule, the digestive powers of persons suffering from diabetes are remarkably good. The stomach and liver are capable of dealing efficiently with the large amount of food submitted to them.

Accordingly *urea* is formed in abundance. It was formerly supposed indeed, that much less urea is contained in the urine of diabetic patients than in that of healthy persons. But it has been shown that this was a mistake, and that the total amount of urea voided in the twenty-four hours is as great as, or even often greater than, the normal quantity. We shall presently see that, at least in some cases of diabetes, a part of the sugar is formed from the nitrogenous elements of the food, or rather from the peptones into which they pass before they are absorbed from the alimentary canal; and it is probable that these peptones, under such circumstances, split up so as to produce two series of substances, of which the one has its final term in sugar and the other in urea. The Rev. Prof. Haughton has given a formula which shows that this may occur in such a way that nearly five grains of sugar should be formed for one grain of urea. We should of course not expect that this proportion should show itself exactly in the urine, since a part of the urea excreted must be derived not from food, but from the nitrogenous tissues, as in health. Dr Dickinson has made calculations, allowing a reasonable proportion for tissue-urea, and he found that in at least one patient the actual daily quantities of urea and sugar varied greatly, the proportion of surplus urea to sugar was almost constantly as 1 to 6·1. On the other hand, Dr Ringer some years ago arrived at the conclusion that during abstinence, or under a non-nitrogenous diet, the *total* amount of urea and that of sugar excreted, whether by different patients or by the same patient at different times, had a constant ratio of about 1 to 2. But his results are difficult of acceptance because they involve the supposition that even the disintegration of the nitrogenous tissues gives rise in diabetes to the formation of sugar as well as of urea.

A further question concerns the amount of *uric acid* which is excreted by diabetic patients. This also was supposed at one time to be greatly less than normal; but Dr Dickinson thinks that the deficiency is probably apparent rather than real. He has seen a copious deposit of crystals of lithic acid in urine of which more than fourteen pints were passed daily. Still in comparison with urea, it appears to be clear that but little uric acid is found in diabetes. It is an interesting fact that, in a disease in which such large quantities of sugar are discharged from the body, unoxidised nitrogenous matters pass off in that form which is the result of their most complete oxidation. And in this connection one of Dr Ringer's results may be noted, namely, that in diabetes the ingestion of *non*-nitrogenous food was followed by a marked increase in the amount of urea, as well as of sugar, in the urine For this may fairly be correlated with the fact pointed out by Murchison that lithæmia in which nitrogenous principles fail to be fully oxidised, is especially apt to be induced by the ingestion of non-nitrogenous matters in excess, or in such quantities that the digestive organs become overloaded with them.

Nor are other facts wanting that appear to show an antagonism between lithæmia and diabetes. Sir Charles Scudamore long ago showed that whereas gout was less common among the Scotch than among the English, the relative liability of the two populations to diabetes was reversed. Dr Pavy mentions that one of his patients, who had before been a martyr to dyspepsia, said that that complaint ceased entirely as soon as he became attacked by diabetes. Recently one of my diabetic patients, who said that he had before been unable to digest cheese, found that in his altered state he could eat large quantities of it without discomfort.

It has also been noticed that gout has ceased to return in persons who have become diabetic: but this is less conclusive because it might be merely the result of the increased flow of urine preventing any accumulation of uric acid in the blood.

These facts, indicating that lithæmia and diabetes are inversely correlated, are interesting not only in themselves but also because they afford further evidence of the correctness of the views which regard both these diseases as disorders of the hepatic functions. No other theories of their origin would account for the existence of a relation between them.

The *bowels* are usually constipated in diabetes, the fæces being dry and hard. But diarrhœa sometimes occurs, and it may lead to a state of prostration which is the immediate cause of death.

Diabetic patients often complain of chilliness, and Dr Dickinson has found that the *temperature* of the body is lowered, bearing some proportion to the severity of the disease. In one very severe case in a boy it varied from 93·6° to 94·8°. When pneumonia set in, which caused death, the thermometer rose, but only to 97·8°. But in another case fatal pneumonia was attended with a temperature of 103·2°. We shall presently see, however, that in many cases the approach of death is preceded not by rise but by fall of temperature.

The *skin* is usually dry and harsh. The cuticle of the palms is stiff, and the furrows have a peculiar white mealy appearance. Generally speaking there is no sensible perspiration through the whole course of the disease, but sometimes profuse perspirations occur. This is difficult of comprehension, and so, perhaps, is the fact that the subcutaneous tissue frequently becomes œdematous. This, however, is no doubt the result of anæmia. When it occurs, the presence of albumen in the urine should be sought for, as Bright's disease is a not infrequent complication of diabetes. But there is often œdema of the ankles, even when the kidneys are healthy. Dr Roberts mentions an instance in which there was ascites and even œdema of the arms and hands.

One of the most marked symptoms of diabetes is *emaciation*. The features acquire a peculiar drawn, pinched look, by which the disease may often be recognised. Sometimes, however, the patient remains well nourished. Dr Roberts says that one of his patients weighed more than fifteen stone, when he had been passing twelve pints of highly saccharine urine for some months; and that one of Prout's patients weighed twenty-three stone.

Another symptom of diabetes is that the patient's breath has a peculiar *sweet smell* like that of the urine but less distinct. Dr Dickinson says that this is connected with a constipated state of the bowels, and he appears to take it as an indication of the near approach of the coma which is often the immediate cause of death. It can be detected by some people even at a little distance. It is said that the late Dr Babington when he came down to take in patients on a Wednesday morning at Guy's Hospital, could tell at once whether there was a case of diabetes among the applicants.

Physiology of glycose.—As I have already stated, the point from which all discussions as to the pathology of diabetes must now necessarily start is the glycogenic function of the liver. Unfortunately opinions are still so unsettled as to this physiological question that I must needs devote a brief space to its consideration. In no other way can I explain what appears to be the theory of the disease.

In 1848 Claude Bernard, of Paris, published the startling discovery that sugar, which had before been supposed to be always a vegetable product, was to be found in abundance in the liver in all animals, even when they had been kept for some time on a diet containing neither sugar nor any of those amylaceous substances that were known to be capable of undergoing conversion into it. He found also that the blood taken from the hepatic vein contained sugar in much greater quantity than that from the portal vein or from the vessels in other parts of the body. From these observations he inferred that sugar was constantly being poured by the liver into the blood, and that it there underwent oxidation, probably with the object of keeping up the animal heat.

Bernard further showed that when the liver had been removed from the body of an animal great quantities of sugar were formed within it. The organ was deprived of all the sugar it contained by the injection of a stream of water through its vessels, and it again became loaded with sugar after being left to itself for twenty-four hours.

It was therefore evident that there is in the liver some substance which is not itself sugar, but which is capable of becoming sugar. And in 1857 this substance was isolated in the form of a white powder. It has been called by various names, among which that of glycogen is the one most generally adopted.

In repeating Bernard's experiments my colleague, Dr Pavy, found that the conclusions drawn from them were inaccurate in one important respect, namely, as regards the presence of actual sugar *during life* in considerable quantity in the liver and in the blood of the hepatic vein. He showed that when certain precautions are taken, by which the tendency of the glycogen to undergo conversion into sugar is arrested at the moment of death, no sugar can be detected in the organ after removal from the body; and by catheterising the right ventricle during life he obtained blood which, although it had come straight from the liver, was free from sugar, or at least contained only a very minute quantity of that substance.

These results of Dr Pavy's seemed at one time to threaten the overthrow of Bernard's views; but they are, in fact, exactly what might have been predicted from a knowledge of the relative properties of sugar and glycogen. The former is a highly diffusible substance, while the latter possesses scarcely any diffusibility. This is why, as in Bernard's original experiment, the sugar can be removed from the liver by a stream of water, while the glycogen remains behind. Now, during life the liver is constantly traversed by a rapid current of blood which must necessarily carry away at once any sugar that is formed in the organ. A little consideration will show that it would not be possible for the liver, and still less for the swiftly flowing blood of the hepatic vein, to contain during life the quantity of sugar which Bernard found in them after death.

On the other hand, it is doubtful whether Dr Pavy's experiments, multiplied and varied as they have been, can be fairly considered to establish the conclusion which he drew from them, namely, that glycogen is not converted into sugar during life, and therefore that some other destination must be discovered for it. The quantity of glycogen present in the liver appears to be always changing; it increases after each meal, and diminishes during fasting. It must therefore be constantly passing into some other substance, or in some other way undergoing removal from the organ. And if we take into consideration the readiness with which it is converted into sugar under the influence of different ferments, we shall certainly require very strong evidence to convince us that this is not its natural destination. A ferment which is capable of effecting its conversion into sugar is, in fact, always present in the blood, and the liver contains a similar ferment, which may be extracted from it by means of glycerine. We shall presently see that an increased flow of blood through the hepatic artery is admitted by all observers to cause a portion of the glycogen to pass into sugar, which is forthwith washed away in the circulation.

Some time ago Dr Lauder Brunton suggested a theory with regard to the glycogenic function of the liver which appears to me to be exceedingly probable. He pointed out that if it were not for this provision the sugar absorbed from the stomach and intestines would circulate in the blood in excessive quantity after every meal. It probably could not be at once made use of by the tissues, and some of it would necessarily be excreted by the kidneys, which drain off from the circulating fluid all diffused substances that are present in it in any considerable quantity. The liver, therefore, takes from the portal blood its sugar, and converts it into the non-diffusible glycogen. This is stored up for a time in the organ. Afterwards, however, during the interval between each meal and the one which succeeds it, the glycogen is gradually reconverted into sugar. The diffusibility of this substance causes it to enter the blood of the hepatic vein, and thence it is carried to all parts of the body.

There may, indeed, be a question whether a part at least of the glycogen does not enter the circulation as such. The white corpuscles of the blood are said to contain this principle; in the exudation of pneumonia, for instance, which is mainly made up of leucocytes, it is present in large quantity. It is therefore possible that the white corpuscles may act as carriers of glycogen, and so diminish the quantity of this substance contained in the liver during fasting. But, however this may be, there is every reason to believe that sooner or later the glycogen always undergoes conversion into sugar.

What purposes are served by this gradual flow of sugar into the blood is still somewhat doubtful. They certainly must be of great importance, for, as I have already mentioned, even carnivorous animals form glycogen from their food. The most modern view is that the sugar undergoes destruction in the blood, lungs, and muscles, and perhaps in other tissues as well. It is supposed, not only to supply the muscles with energy for their work, but also to maintain by its oxidation the temperature of the body.

Pathology.—We must now consider what light can be thrown upon the nature of diabetes by means of these physiological facts and theories. And in the first place it is to be noted that the presence of sugar in the urine of a human being is not always by itself a proof that he has diabetes. This substance is occasionally excreted in considerable quantity by the kidneys of persons who are in good health. Generally speaking, this is quite transitory; it is, indeed, an accidental occurrence.

Thus, when a person has inhaled chloroform or ether, his urine may give a reaction with the copper test for a short time afterwards. It is true that chloroform itself possesses the property of reducing the oxide of copper, and its excretion by the kidneys is not at all improbable; but Dr Pavy and others believe that this is not the cause of the reaction which the urine gives under such circumstances, and that sugar is really present. Then, again, the urine has been found to be saccharine after recovery from cholera and after croup; and a paroxysm of whooping-cough, asthma, or epilepsy has been known to have the same effect. It is also said that sugar has been detected in the urine after the ingestion of a very large quantity of saccharine or amylaceous food.

Now, for all such cases the term diabetes is obviously inapplicable. We have another name, glycosuria (*i.e.* glycose in the urine), which should always be used for them. They can all be explained without difficulty on physiological grounds. Thus, in animals, when sugar is injected into the portal vein too rapidly for the liver to convert it all into glycogen, a portion of it passes into the general circulation and appears as glycose in the urine. Again, Dr Pavy found in all his experiments that whenever an animal struggled the quantity of sugar in the blood of the vena cava and carotid arteries was for the time increased. It is probable that the urine would be found to be more or less saccharine in a great variety of pathological conditions in which the circulation through the liver is disturbed, were it not that in disease the amount of glycogen stored up in the organ is too greatly reduced for the production of these forms of transitory glycosuria.

From an etymological point of view, glycosuria would appear to be a better name than diabetes for another class of cases, in which, again, all the ordinary symptoms of diabetes are absent, but in which, instead of being transitory, the presence of sugar in the urine is persistent. I shall presently describe some of these forms of the disease in detail, but I may now say that I believe there is in practice no definite boundary line between such cases and those in which the most marked symptoms are present. Indeed, the same patient may come in turn under the one and under the other category. I find that the same treatment is beneficial in both kinds of cases, and I think that the same name should be given to both; that, in fact, persistent glycosuria is always diabetes.

It is evident that there are three principal ways in which it is conceivable that the natural processes might become perverted so as to lead to the excretion of sugar by the kidneys. These are:—1. The entrance of sugar into the general circulation, without its having been arrested in the liver and converted into glycogen; 2. An excessive flow of sugar from the liver into the blood; and 3. A diminished destruction of sugar in the blood. Now, Dr Brunton ('Reynolds' System,' vol. v, p. 399) admits all these as causes of diabetes, and divides them again into minor heads. To me, however, it appears in the highest degree improbable that this disease should in turn arise in each of all the different ways in which it can possibly be imagined to be produced. And I find good reasons for believing that it is always due to the second of the three causes which I have enumerated, viz. to an excessive flow of sugar from the liver into the blood.

Let us, as the *first* theory, consider whether diabetes can ever be fairly attributed to a mere failure in the glycogenic function of the liver, allowing a crude form of sugar to enter the general circulation. It has, in fact, been proved experimentally that unless it has first passed through the liver the sugar absorbed from the food is incapable of being utilised in the system, and that it is of necessity excreted by the kidneys. And we can easily conceive that the same thing may occur as the result of disease. But it is to be observed at once that such a theory can be applied to very few instances of diabetes, as we see it clinically. In all severe cases of this disease the urine still remains saccharine, even when the patient's diet is restricted, so that it contains neither sugar nor any of those principles which are commonly regarded as being convertible into sugar. Under such circumstances there can be no doubt that the sugar is formed by the liver, just as in healthy carnivorous animals it is formed by that organ out of the other elements of the food. But there are other cases in which it certainly appears at first sight to be possible and even likely that the disease may be due to a defective glycogenesis on the part of the liver, namely, those in which the urine ceases to contain sugar as soon as the patient confines himself to a properly restricted diet. Nay, since we have seen that in other cases the liver not only can, but does, form sugar out of food containing neither sugar nor starch, it might be thought that for these cases such a theory is not only tenable, but is the only one that can be held.

Thus, Dr Bence Jones is said to have declared that in half the cases of diabetes that came under his observation the disease consisted simply in an arrest of change in the food sugar, and Dr Pavy has expressed a similar view with regard to the cases now under consideration.

But a closer examination of the question cannot fail, as I think, to suggest doubts as to the validity of this explanation. If such a patient, whose urine is kept free from sugar by a restricted diet, one day breaks through the dietetic rules laid down for his guidance and eats an apple or drinks a glass of sweet beer, it will generally be found that the secretion again becomes saccharine. This is no more than might be expected; but now comes a circumstance which is remarkable, and for which some further explanation is evidently necessary. The quantity of sugar that is voided is altogether disproportionate to the amount contained in the offending article; and sugar often continues to be excreted for a very long time afterwards. Dr Pavy mentions the case of a patient in whom the disease had been kept under control by strict dieting, and who drank about a pint of cider. His urine thereupon became loaded with sugar, and remained so for a period of two months, before it again became normal. Now, it appears to me that the only hypothesis which is capable of explaining such facts as these is that the saccharine or amylaceous article of food exerts some directly injurious influence, causing the blood to contain an excess of sugar for a considerable period afterwards. And if we believe that the liver is the organ principally concerned in supplying the blood with sugar we can hardly help inferring that this injurious influence is exerted upon the liver. It seems as if saccharine food were a *poison* to a patient who is affected in this way. But it is evident that this very hypothesis affords an adequate explanation also of the fact with which I started, namely, that in certain cases of diabetes the urine contains sugar only so long as the patient takes saccharine or starchy matters in his food.

We may next consider whether there is reason to believe that diabetes is ever due to the *third* of the causes that I enumerated above, namely, a deficient destruction of the sugar in the blood.

Now, Dr Owen Rees formerly showed that the sugar which is found in the urine in diabetes does not possess exactly the same properties as the sugar which is formed from glycogen in the liver under normal conditions. It is much less ready to undergo destruction in the presence of blood or other animal matters. It might, therefore, be thought that, even in the most extreme case of diabetes, the total amount of sugar formed during the twenty-four hours might not really be excessive, its escape in such large quantity in the urine being due to its failing to become used up by the muscles, lungs, and other tissues. It must be borne in mind that we have no knowledge as to the absolute quantity of glycogen which is naturally formed by the liver each day and subsequently converted into sugar.

But if the physiological theory that I have given above is correct, there is a test which may be applied without difficulty to any case which is supposed to be due to this cause. We have seen that the object of the glycogenic function of the liver is to equalise the flow of sugar into the blood, instead of its entering the circulation in large quantity after each meal and being absent during fasting. We have therefore reason for expecting that in any case of diabetes due to imperfect destruction of sugar, the liver performing its functions naturally, the excretion of sugar should be uniform throughout the day, and that it should be entirely uninfluenced by the meals. Now, it appears that this is never the case. At any rate I believe that when the amount of sugar voided in the urine by a diabetic patient at different periods of the day has been determined great variations have always been found. In illustration of this I may refer to a case which Dr Pavy studied very carefully (and of which he has published a detailed account in his work). A man, whose name was Joseph North, was placed in succession upon different kinds of food, and his urine was collected and analysed every four hours. As a very general rule, the amount of sugar excreted in the urine was considerably greater between 5 and 9 p.m than at any other part of the day; while, on the other hand, it was commonly at its lowest point during the hours of the night and early morning. These variations were evidently due to the influence of the food taken during the day. A striking illustration of this is afforded by one occasion when the man departed from the instructions given to him, and at 4 p.m. drank some cocoa sweetened with sugar. Between 5 and 9 p.m. of that day his urine contained 1311 grains of sugar, whereas in the twenty-four hours previously the quantity in equal periods of time had ranged between 166 and 468 grains; and from 9 to 1 a.m., after the cocoa had been taken, it was again only 483 grains.

Now, for such a case as this it is evident that any theory which should refer the diabetes simply to deficient destruction of sugar would be altogether untenable. And, as I have already stated, we have at present no reason for doubting that similar variations in the amount of sugar excreted at different periods of the day occur in all cases of diabetes.

We will conclude, then, that diabetes or persistent glycosuria is always due to the *second* of the three causes which I have enumerated on p. 415, namely, to an excessive flow of sugar from the liver into the blood.

Cause of increased glycogenesis.—It remains to determine how the excessive flow is brought about. The well-known discovery of Claude Bernard was that in the lower animals the operation of puncturing a spot in the floor of the fourth ventricle is followed by glycosuria, or (as it is called) by artificial diabetes. No fact in experimental pathology is more firmly established than this. And the explanation of it which is at present accepted is that the puncture irritates the roots of the vagus nerves, and so "inhibits" the vaso-motor centre of the nerves distributed to the hepatic blood-vessels. The vaso-motor nerves being thus paralysed, the hepatic artery dilates. The flow of blood through that vessel is thereby augmented, and the result is an increased conversion of glycogen into sugar, and a flow of sugar into the blood in excess of the requirements of the system.* Whenever the liver of an animal happens to contain no glycogen, the operation fails to cause an excretion of sugar in the urine.

Now, this explanation substitutes for the *excessive formation of sugar* which has been shown to be the cause of ordinary diabetes a mere *increased rapidity in the discharge of sugar* into the blood. And it is important to observe that such a view, although it may account for the presence of sugar in the urine in Bernard's experiments, will not explain the disease. For, according to what has been stated, it is clear that after the puncture, in proportion as the amount of glycogen in the liver becomes reduced by the excessive demands made upon it, the glycosuria must pass off; and, in fact, after a few hours sugar can no longer be detected in the urine of animals on which this experiment has been performed. It is true that Schiff found in rats that by dividing certain columns of the cord he could make the animals diabetic, and that this condition would last for two or three weeks. But I cannot think that even these results warrant our attributing ordinary persistent diabetes in the human subject to paralysis of the vaso-motor nerves. It seems to be impossible that the excretion of such large quantities of sugar as are passed by patients affected with this disease, at all hours of the day, and in amounts influenced only partially by the meals, can be due to a mere increased rapidity of the conversion into sugar of the glycogen naturally formed by the liver.

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* Dr Pavy has shown that one can bring about the same result experimentally by causing arterial blood to flow through the portal vein. He draws the conclusion that an arterialised state of the blood in this vessel is the actual exciting cause of diabetes in the human subject; but for this hypothesis there does not appear to be sufficient foundation.

It is true that this difficulty might be removed by introducing, as an auxiliary cause, Dr Rees' observation, that diabetic sugar possesses greater power of resisting oxidation than the sugar which is normally formed by the liver. As I have already shown, this cannot be the sole cause of diabetes. But the line of reasoning which I have adopted at p. 416 would be no bar to the acceptance of this as a second factor in the development of the disease, if it were also assumed that, in consequence of the vaso-motor paralysis, the flow of sugar into the blood from the liver is accelerated after each meal, instead of being equable as it is in health.

But it appears to me much more probable that the really important second factor in the causation of diabetes (in addition to an increased rapidity in the conversion of glycogen into sugar and its discharge into the blood) is an *excessive production of glycogen* itself by the liver. I have little doubt that the exalted activity of the glycogenic function of the organ—which we have seen to occur so far as the conversion of glycogen into sugar is concerned—occurs likewise in regard to the other process which belongs to that function, namely, the formation of glycogen from the various matters which are brought to the liver in the blood; including products of disintegration of the tissues, as well as substances ingested and absorbed by the walls of the stomach and intestines.

That this is the case seems, in fact, to be established by Dr Ringer's observations already referred to (p. 411), that in diabetes the ingestion of non-nitrogenous food is followed by an increase in the secretion of urea. This can only be explained by the supposition that the sugar which is taken with the food stimulates the liver to destroy more proteid matter than in health.

Diabetes, then, appears to be the result of an excessive activity of the glycogenic function of the liver. In the first place an undue quantity of glycogen is formed from the materials brought to the organ. In the second place, this glycogen is converted into sugar more rapidly than under normal conditions. It also seems that the sugar which is formed in diabetes has a greater power of resisting destruction in the blood than the sugar which is produced in the liver in a state of health. Upon the degree to which the natural processes are perverted depends the severity of the disease.

Nervous origin.—These considerations render it improbable that true diabetes is caused by disease of the nervous centres acting upon the liver through the vaso-motor nerves, as in the affection artificially induced by Bernard and others. Many very interesting cases have indeed been recorded in which the urine has been found to contain sugar after the brain has been injured, or when that organ has been the subject of disease. As far back as 1854, Dr Goolden published a series of instances of glycosuria following blows or falls upon the head. But in most of them the presence of sugar in the urine was transitory, so that they would hardly be regarded as examples of diabetes. Dr Pavy, however, mentions the case of a cadet at Woolwich who was attacked with strongly marked diabetes a few days after being stunned by a violent blow on the head.

In certain cases, again, common forms of cerebral disease have been followed by diabetes. Dr Pavy mentions two instances in which this occurred after an attack of hemiplegia, but the patients were old men, in whom the urine is comparatively apt to become saccharine, so that it may have been a mere coincidence. The most striking case in which disease in the neighbourhood of the fourth ventricle has led to a saccharine state of the urine in man appears to be one referred to by Trousseau, in which there was a tumour in the floor of this cavity. Moreover, the same writer describes two other cases of ordinary diabetes, in each of which the same part was found to be hyperæmic, and to present scattered spots of a tawny brownish colour, in which the cells of the nervous tissue were degenerated. Among English pathologists, Dr Dickinson is, I think, alone in having detected morbid changes in the pons Varolii or neighbouring parts of the brain in diabetes. He has described eleven cases occurring consecutively, in all of which he found degenerative changes in the cerebral substance surrounding the smaller arteries in different parts of the encephalon and spinal cord, but especially in the medulla oblongata and the pons. The earliest alteration appeared to him to be a dilatation of the vessels and often an extravasation of their contents. This was followed by a destruction of the adjacent tissue, so that cavities were formed, often large enough to be distinctly visible to the naked eye, and containing, beside the vessels, extravasated blood, pigment-granules, and the products of the decay of nervous matter. The nerve-cells were perfect, and, indeed, the degenerative process was generally more marked in the white than in the grey substance, although it was particularly distinct in the floor of the fourth ventricle.

Ætiology.—The causes of diabetes are as yet very imperfectly understood; our knowledge on this head is fragmentary. Sometimes diabetes appears to be a direct result of mental anxiety or overwork. Dr Herman Weber met with the case of a gentleman who became diabetic on two separate occasions, at an interval of nine years, under the pressure of intense anxiety from impending commercial ruin. He recovered completely from the second attack, and lived four years longer, to die of apoplexy. In other cases it has followed a violent fit of grief or of anger. Prout thought that it might be caused by exposure to cold, or be the product of rheumatic attacks, themselves brought on by cold and moisture, but this appears extremely doubtful.

Whatever may be the immediate exciting cause of diabetes, an inherited tendency is certainly sometimes a factor in its causation. Dr Pavy gives numerous instances of this. In one family two sisters and two brothers; in another a son, his father, and an aunt; in a third a father and his two daughters, in a fourth a father and a son; in a fifth two brothers; in a sixth three brothers; in a seventh a brother and a sister; and in an eighth a mother and a sister—all perished of this disease. Dr Roberts speaks of a family of eight children, all of whom became diabetic, although the parents were healthy.*

Prout long ago remarked that people with red hair were particularly liable to the disease; and I believe that it is so.

The period of life at which diabetes is most apt to occur is that of sexual maturity. It rarely attacks children under five years of age. I do not know of any other instance occurring at so early a period of life as one observed by Dr Brown in a child twenty months old, who died before completing her second year, of miliary tuberculosis. According to Dr Roberts females become less liable to it after forty-five years of age; whereas in males there is no decrease in the mortality from diabetes until the age of sixty-five years has been passed. It is far more common in men than in women (in cases at Guy's Hospital above two to one). Dr Dickinson says that more numerous cases occur in agricultural than in manufacturing districts; whereas Dr Roberts has made exactly the opposite statement.

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* [Mr J. E. Nevins has collected for me 122 cases from the records of Guy's Hospital, and in eight of these there was a history of diabetes in one or more members of the patient's family. Adding cases obtained by the courtesy of the registrars of the three other large hospitals, St Bartholomew's, St Thomas's, and the London, and those recorded by Schmitz and Griesinger, he finds thirty-five hereditary cases in a total of 537. In one case the mother and seven of the mother's brothers had died of diabetes.—Ed.]

Exceptional cases.—I have already remarked that in some cases of diabetes the ordinary symptoms are absent; and I must now call the reader's attention to the anomalous forms of disease in which this occurs.

The least common circumstance is perhaps for sugar to be discovered in the urine of persons who believe themselves to be perfectly well. Of this Bence Jones gave an instance. A gentleman noticed some small masses in his urine, and consequently had it tested. They proved to consist of epithelium from the bladder, but there was sugar in the urine, and this continued to be the case whenever it was examined afterwards. He was a stout man, and remained in good health. Another case, mentioned by Griesinger, is that of a medical student, whose urine was saccharine during the whole of one winter, while he was residing in a moist and foggy locality in Switzerland. He never had a single symptom of diabetes. Before and afterwards the urine was often tested and found normal.

Instances such as these, however, appear to be of quite exceptional occurrence. In the immense majority of cases in which sugar is found in the urine of an individual who does not present any of the ordinary signs of diabetes, he nevertheless is far from being well, and complains of more or less definite symptoms of some other kind. Most commonly he suffers from one or another of a limited number of affections, which may indeed occur in patients in whom the urine is normal, but which nevertheless are so often accompanied by glycosuria that no cautious practitioner ever deals with them without ascertaining whether this is or is not present. The affections in question are perhaps best described as "complications" of diabetes; but no account of the symptoms of this disease can be regarded as complete in which they are not mentioned. The recognition of the saccharine condition of the urine is of infinite importance in all such cases, because it is very often the key to their successful treatment.

One of these affections is *pruritus vulvæ* in women, often attended with a lichenous or eczematous eruption. That such complaints are often due to diabetes is mentioned by most writers; and I have recently seen two very striking instances. One occurred in a lady, aged fifty-two, who came to me on account of an eruption affecting the vulva, and attended with severe pruritus. The parts were reddened, but dry; the disease in fact resembled a chronic lichen. When I first saw her I had not an opportunity of examining the urine; and my prescription did her no good. I told her to bring her water next time; and it contained ten grains of sugar to the ounce. In a few weeks, under proper treatment, her troublesome complaint was removed; and this although the urine still contained a small quantity of sugar. I do not know that ordinary eczema of the vulva, with profuse discharge, is ever caused by diabetes; in all the cases that I have seen, the eruption has been dry. Most writers say that its cause is the local irritation set up by the sugar in the urine; and they remark that the orifice of the male urethra, and the glans penis are sometimes excoriated under the same circumstances. But in opposition to this view it must be said that lichen and pruritus of the vulva comparatively seldom occur in those cases in which the quantity of urine is very great, and in which the typical symptoms of diabetes are fully developed.

Again, *carbuncles* and *boils* are apt to arise in patients whose urine is saccharine. Dr Prout says that in his experience diabetes always accompanied carbuncles and malignant boils or abscesses. But it has since been shown that this is too sweeping a statement. The importance of remembering the liability of diabetic patients to carbuncular affections is well shown by a case of Sir William Gull's which is related by Dr Pavy. A medical man was suffering from cerebral symptoms, for which it was suggested that he should apply a blister to the nape of the neck. His urine contained sugar, and on this account he has cautioned against adopting such a treatment. However, the blister was employed; and a large carbuncle soon developed itself which proved fatal. On the other hand, Dr Prout speaks of glycosuria as a temporary accompaniment of affections of this kind. A patient of his, middle-aged, told him that for a long period he had been subject, at intervals of a year or two, to boils and carbuncles, and that during such attacks he always passed a quantity of saccharine urine, whereas at other times the secretion was natural. Later writers also have given cases in which patients have had sugar in the urine only while they suffered under some carbuncular affection.

Gangrene of one of the lower limbs, resembling senile gangrene, is also frequently associated with a saccharine state of the urine. This is a fact which has been especially insisted upon by the surgeons of Dublin; and several cases of the kind have occurred in Guy's Hospital.

Defective sight is another symptom which is common in diabetic patients. Most frequently this is due to impairment of the power of the ciliary muscle; and in such cases Dr Pavy has found that the application of Calabar bean to the conjunctiva is very beneficial. Sometimes, however, the loss of sight is due to the formation of *cataract.* Some years ago Mr France published several cases of this kind in the 'Guy's Hospital Reports.' Diabetic cataract has acquired special interest from certain experiments, made by Dr Weir Mitchell, in which frogs were immersed in a saccharine solution, or had such a solution injected into the cellular tissue, with the result that the crystalline lens became opaque. But Dr Roberts thinks that this is probably a mere coincidence, however unlikely such a coincidence may appear. He insists on the fact that the frog's lens remains opaque only while the animal is immersed in the saccharine liquid; whereas in man the cataract is permanent. Moreover, diabetic cataract is often for a time confined to one eye. Another cause of loss of sight in diabetes is atrophy of the optic discs; of this I have myself seen an instance.

Another frequent complication is a destructive disease of one or both lungs, which is very like that found in the more acute forms of *phthisis.* In comparison with the other affections of which I have been speaking this is of vast importance, for it is very often the immediate cause of the patient's death. I have before me notes of all the cases in which diabetes has led to a fatal termination in Guy's Hospital during the last twenty years; they are forty in number, and in seventeen of them the immediate cause of death was phthisis. Now, Dr Addison taught, and Dr Wilks has since maintained the same opinion, that the pulmonary disease in cases of this kind is not a tubercular phthisis, but rather a form of pneumonia. Some writers have hesitated to adopt this opinion. I have therefore carefully searched our records to see how far they support Dr Addison's view, and I find that in twelve among the seventeen cases there was nothing that could fairly be identified as tubercle in the lungs, and in all of these it is either expressly stated that the larynx and intestines presented no tubercular ulceration, or, at least, no mention is made of these organs. On the other hand, it is said that in four cases the lungs contained grey or miliary tubercles; and in two of them, as well as in the other case, the intestines showed tuberculous ulcers. Now, referring the reader to my first volume for a discussion of the relation of "pneumonic" phthisis to the ordinary "tubercular" disease (p. 926), I may say that such a proportion of cases without tubercles in the larynx and intestines is, according to my experience, very different from what occurs in any form of phthisis, apart from diabetes: hence it supports very strongly the opinion that the pulmonary affection in this disease is not of a tubercular origin. It might, indeed, be urged that the development of mischief in the lungs is so rapid that there is no time for the formation of tubercles in other parts of the body: in several cases the earliest pulmonary symptoms appeared only from two to five months before the patient's death. But that this is not the only reason why the intestines and larynx escape, is evident from one case in which the symptoms of disease of the lungs preceded those of diabetes, and began fourteen months before death: for even in this case the intestines were free from tubercle. The pulmonary affection generally spreads through the lungs from apex to base, like ordinary phthisis; it very rapidly leads to the formation of one or more large cavities, by which the whole of an upper lobe may be excavated, and which have usually very thin ragged walls. It is generally much more advanced in one lung than in the other, but sometimes attacks both organs pretty equally. In further proof of the rapidity with which it advances to a fatal termination, I may observe that in only one of the twenty-eight cases in which death was not the direct result of this form of pulmonary disease, did the lungs present any trace of it. If it were ordinary phthisis, we may be sure that it would be commonly found, in an early stage, in those cases in which the patient dies from other causes.

Another frequent cause of death in diabetes is ordinary lobar or croupous *pneumonia*. This was present in ten out of the forty cases which I have collected from the post-mortem records of Guy's Hospital. In four of them the hepatized parts were more or less distinctly passing into a gangrenous state. The onset of pulmonary symptoms in these cases was generally well marked, and occurred two or three days before death.

Of the remaining fatal cases there were six in which the immediate cause of death was the supervention of *cerebral symptoms*. These generally began with drowsiness, and in a few hours passed into coma. Once or twice there was more or less well-marked delirium, or even convulsions; the pulse was often very feeble, and the temperature low. Indeed, in the cases of this kind which I have seen the state of the patient has been one of collapse quite as much as of coma. In the 'Guy's Hospital Reports' for 1873—74 I recorded one such instance, in which the pulse was scarcely susceptible, and the body and limbs were cold. In this case twenty-six ounces of a solution of phosphate of soda and chloride of sodium, of sp. gr. 1020, were injected into the right cephalic vein, with the effect of restoring the patient to consciousness for a time. He sat up, answered questions, took nourishment well, and even asked for it; his pulse was 80. Thirty-two hours later, however, he again became drowsy and died. This patient's condition, before the solution was injected into his veins, was strikingly like that of a man in the collapse of cholera. Only a few drops of thick dark blood escaped from the wound in the arm. A few months afterwards my colleague, Dr Frederick Taylor, had a similar case, in which he employed the same treatment, but with scarcely any good result (ibid., vol. xix, p. 521).*

In addition to the six cases I have mentioned, in which death was preceded by more or less marked cerebral symptoms, there were two in which it was quite sudden. Probably these should be placed in the same category with the others. There is one peculiarity with regard to a large proportion of these eight cases, namely, that the fatal symptoms developed themselves very shortly after the admission of the patient into the hospital. In five of them death took place within five days from the date of admission, and in three of them it occurred either on the day of admission itself or on the following day. The cause of the sudden fatal termination was, no doubt, the fatigue and excitement which the patients underwent in coming to the hospital. Exactly the same thing was noticed long ago by Dr Prout, who says that four of his private patients sank almost immediately after coming to London from the country to consult him, and one of them was very near dying in Dr Prout's own house. On the other hand, Dr Pavy says that those cases of diabetes in which the disease has been partially kept under control by treatment are particularly apt to terminate fatally by coma; whereas when the disease is allowed to run on unchecked, the chances are in favour of the supervention of pulmonary disease. With regard to this I may observe that my recent experience in the post-mortem room has shown that great caution is necessary in assigning the cause of death in diabetes, unless an autopsy is made. In each of the last two cases of diabetes that have terminated fatally in Guy's Hospital the patient died with cerebral symptoms, but in each of them death was found to be due to a form of local inflammation. In one the pelves of the kidneys were dilated and inflamed, and the tissue of one kidney presented numerous points of suppuration. On inquiry I found that the patient had complained greatly of pains in the loins for some days before death. In the other case there was extensive pneumonic consolidation of the base of the left lung.

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* [This "diabetic coma," ably described by Prof. Kussmaul, of Strassburg, has been ascribed by Petters to the presence of *acetone* in the blood. But acetonæmia is not always present in diabetes, nor is it always, when present, accompanied by cerebral symptoms. See an excellent account of Acetonæmia and Lipæmia in Diabetes, in Prof. Gamgee's 'Physiological Chemistry,' pp. 168—172.—Ed.]

Morbid anatomy.—The inflammation of the kidneys in the former of these two cases was so exactly like what occurs in cases of stricture and other diseases of the urethra or bladder that these parts were very carefully examined. The urethra was perfectly healthy; the *bladder*, on the other hand, was greatly hypertrophied. This led me to consider whether the increased thickness of the coats of this viscus could be due to the augmented work it had had to perform in consequence of the over-secretion of urine. In the second case, therefore, I looked at the bladder with much interest, and found that it also was markedly hypertrophied, and that its mucous coat protruded between the muscular fasciculi, so as to form numerous sacculi. I think that it is probable that hypertrophy of the bladder may be found to be constantly (or, at least, very frequently) present in diabetes. In one case published by Professor Haughton in the 'Dublin Medical Journal' for 1861 it is incidentally mentioned that the bladder was in this condition, although no stress is laid upon the fact.

With regard to the other appearances found on post-mortem examination of those who have died of diabetes, there is very little to be said beyond what has already been incidentally mentioned. The viscera have a very decided sweet smell, resembling that observable during life in the urine and breath; and it may be noted that in one case Dr Wilks found that this odour was still observable, although the patient died of typhus fever and pneumonia, and his urine (which also retained the sweet smell) had been free from sugar for some days before death. The liver and kidneys are sometimes large. Dr Wilks thought the appearance of the *liver* differed somewhat from that presented by the organ under other circumstances; he described it as having a uniform fleshy appearance, resembling that of boiled beef; but I cannot myself confirm this. The *kidneys* are not infrequently large, soft, and fatty; and they are sometimes affected with one or other form of morbus Brightii. According to Niemeyer the pancreas is sometimes hypertrophied.*

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* [In some cases of diabetes the blood has been found creamy from the presence of fatty molecules, and fat-embolism has been found in the lungs (Sanders and Hamilton,

Prognosis.—In its well-marked forms diabetes always tends to terminate in the death of the patient within a comparatively short period of time. It is true that one cannot often state definitely what is the whole duration of the disease, because its onset is generally insidious; but for practical purposes this is of no consequence ; the important fact is that when the urine has once been found to be persistently saccharine, the days of the patient are numbered, unless by treatment the progress of the malady can be arrested. Cases are sometimes met with, in which death occurs within a few weeks from the time when the first symptoms of the disease show themselves. Dr Paget even observed a case in which the patient, a Cambridge undergraduate, supposed to be in perfect health, took part in athletic sports, and actually came in second in a running race, within twelve days of his death. More frequently, however, the duration of diabetes is from one to three years. Dr Prout said in 1848 that, among nearly seven hundred patients whom he had seen within thirty years, he then knew of but two in whom the disease had been perfectly developed ten years before. Dr Dickinson mentions the case of one patient in whom the urine was constantly saccharine for fifteen years.

The *age* of the patient appears to have a greater influence than any other condition on the rate with which diabetes progresses towards a fatal termination. Children who are attacked by it never live to grow up. On the other hand, old people sometimes pass saccharine urine for years without appearing to suffer much in health. In such patients, however, the urine is seldom excessive in quantity, nor are marked symptoms of diabetes generally present. At a middle period of life the prognosis of a case of diabetes, when it first comes under observation, must mainly be based upon the degree of severity of the symptoms. Thus all writers mention that it is a very favourable sign if the patient remains well nourished and florid. It is generally said that the detection of albumen in the urine in addition to the sugar is a serious indication, but Dr Pavy says that he has known a small quantity of albumen to be present for years without any harm arising to the patient.

But these considerations, which guide the physician in forming his first impression as to the probable issue of a case of diabetes, require to be combined with, and corrected by, the results of treatment, before a final judgment is pronounced. For medical science sometimes attains more brilliant results in this disease than perhaps in any other of equally dangerous tendencies, and the cases which do best under treatment are by no means always those which appeared at first sight the most promising.

Treatment by diet.—If the view which I have taken of the pathology of diabetes be correct, the rational treatment of the disease is clearly to limit as far as possible the ingestion of any substances which experience shows to be especially apt to stimulate the liver to the excessive formation of glycogen and sugar ; and as I have already stated, all starchy and saccharine matters have this property. Thus, as Dr Prout long ago said, "diet is the first and chief point to be attended to." John Rollo, towards the end of the last century, appears to have been the first to make a suggestion of this kind; and he proposed to confine diabetic patients entirely to animal food. But although the inhabitants of arctic regions and the trappers of North America go for months together without the chance of obtaining any vegetable aliment, experience shows that in civilized life, when all kinds of food are within reach, there is very great difficulty in keeping patients exclusively on meat; and the more so, since in diabetes the appetite is voracious, and the craving for forbidden food all the greater. Of late years, therefore, all those who have studied the subject have striven to include

'Edin. Med. Journ.,' July, 1879). But this condition of the blood is far from constant and is probably very rare.—Ed.]

among permitted viands as many of vegetable origin as possible. The result has been the construction of a tolerably copious diet-table.

Almost all kinds of *animal food*, flesh, fowl, and fish, may be eaten by persons suffering from diabetes. But it is necessary to bear in mind that the ordinary methods of cooking such food often introduce deleterious matters. Thus, soups are thickened with farinaceous matters, jellies are sweetened, even roasted joints are basted with flour. There are few alimentary substances of animal origin which are themselves noxious; but such is the liver of calves or pigs. Such are perhaps the edible molluscs, oysters, cockles, mussels. All of these undoubtedly contain much glycogen, and Dr Roberts forbids them; but it may be noted that in the case of Joseph North, to whom Dr Pavy twice gave four dozen of oysters for two days running, they caused no decided increase in the amount of sugar excreted. *Honey* is of course injurious; and so is also *milk*, but not in the degree that might have been inferred from the amount of sugar contained in it. Dr Pavy indeed found that in North's case the administration of three pints of milk daily not only caused a marked increase in the amount of sugar, but also brought back the uneasy sensations which the man had experienced when the disease had been uncontrolled by treatment. But Dr Roberts mentions the case of a girl who (her diet being restricted in other respects) continued to gain strength and to improve in health when she was allowed to drink three pints of milk daily.

Cream and butter form important articles of diet in diabetes. Cod-liver oil is useful in a similar way. Glycerine might fairly be expected to be harmless, since its composition is so different from that of sugar. Dr Pavy, however, found that it caused a great increase in the sugar excreted; but then the daily quantity which he administered was from six to ten ounces, and perhaps it does not follow that smaller quantities would be injurious.

This is the proper place to mention Dr Donkin's plan of treating diabetes by restricting the patient entirely to a diet of *skimmed milk*, of which he gives six or eight pints daily. The success which its inventor obtained with this mode of treatment is somewhat difficult of explanation, if his results are compared with those of others. Dr Roberts refers to the cases of several persons treated with skimmed milk, and says that few of them could tolerate it for more than a few days, and then were rapidly reduced. Under this method one case was brought to a fatal termination in three months, although a fair state of health had been maintained for a considerable time before. Dr Greenhow, however, has recorded a case in which a patient whose habits had been very intemperate, took from four to six quarts of skimmed milk daily with the effect of entirely removing his symptoms and of freeing his urine from sugar. Two months afterwards he remained still quite well.

On the other hand, most *vegetables* are injurious to patients affected with diabetes. The most noxious of all are said to be potatoes; and carrots, parsnips, turnips, peas, beans, Brussels sprouts, cauliflower, broccoli, asparagus, and sea-kale are also to be forbidden.

Greens, however, as well as spinach, lettuces, watercresses, and mustard and cress, may be eaten; and even small quantities of radishes and celery. The general rule is that all white parts of vegetables, in which chlorophyll has not been developed by exposure to sunlight contain sugar, and are harmful. But by boiling in a large quantity of water, even the forbidden kinds of vegetable, if they contain sugar only and not starch, may be rendered much less injurious. According to Dr Prout, there is a direct advantage in the use of such green vegetables as are harmless; for they are in great part incapable of being digested, and so tend to correct the constipation which is often so troublesome in diabetes.

All kinds of sweet fruits appear to be injurious, although it has been said that currants and raspberries and other acid fruits may be eaten by diabetic patients. Nuts are harmless; but chestnuts abound in starch.

Wheaten flour, as well as that of other kinds of corn, is of course very injurious; and so are rice, arrowroot, sago, tapioca, and even maccaroni and vermicelli. It is with regard to *bread* that the diabetic patient finds the greatest difficulty in carrying out the rules of diet laid down for him. Consequently, there have been many attempts to invent a substitute for bread, which forms so large a part of the food taken by most civilised races of man. The earliest of these attempts was perhaps that of Bouchardat, who, in 1841, suggested that a kind of bread should be made from the gluten of flour, after removal of all the starch by washing. But this gluten bread is by no means a perfect article, for it generally contains a good deal of starch, so that it at once turns blue if a little iodine be dropped on it. Patients also complain that it is very unpleasant to chew, feeling like india-rubber between the teeth. Dr Pavy says that gluten biscuits are much more palatable, but these will not keep more than ten days. Gluten is also prepared as a kind of flour, and in many other ways, to be used for cooking purposes, by which a variety may be given to the meals laid before a diabetic patient. A second substitute for ordinary bread is a sort of cake made from bran. Dr Prout appears to have first suggested this, and Dr Camplin, who was himself affected with diabetes, greatly improved the method by which it is prepared. Bran cakes are still made according to Dr Camplin's directions. A third substance which may be used by diabetic patients instead of bread is the almond food which was introduced by Dr Pavy; its chief fault is its comparatively high price.

It is of course necessary to regulate the *drink* as well as the food of diabetic patients. And the first question is whether the quantity of fluid taken into the stomach should be restricted. This, it appears, is attended with but little advantage, and it adds greatly to the patient's discomfort. The beverages which are admissible are tea, coffee, cocoa from nibs, dry sherry, claret, burgundy, chablis, hock; also brandy and other unsweetened spirits, Burton bitter ale in moderation, and, of course, soda water. Chocolate, sweet ales, porter and stout, cider, all sweet and sparkling wines are to be forbidden. It is not desirable that alcohol should be taken in any considerable quantity by diabetic patients.

It remains to be considered whether the restricted diet should be commenced gradually or suddenly. Most writers advise the former, but I have generally seen the latter plan adopted. On the principle laid down above of the *poisonous* action upon diabetic patients of saccharine and starchy substances, the effects of which persist for a considerable time after their ingestion, the excretion of sugar in the urine cannot be expected to subside otherwise than gradually even when all such substances have been withdrawn from the food, and experience shows that this is the case. Dr Pavy says that the patient often at first complains that his food is not bulky enough to satisfy him; but after persevering for a few days he finds that his appetite becomes less. Johnson's dictum that it is easier to abstain than to be abstemious is very applicable to diabetes.

Before a patient suffering from this disease adopts the restricted diet, and also while it is being carried out, the amount of urine passed each day and its specific gravity should be carefully registered, and the quantity of sugar should also be accurately determined at frequent intervals, if not every day. In no other way can the progress of the case be properly watched, or an opinion be formed as to the necessity for subsidiary treatment. Moreover, a daily analysis enables the physician to check the proceedings of his patients, and to tell with considerable certainty whether he is adhering strictly to the prescribed rules. Those who suffer from diabetes are often untruthful, and wilfully deceive their medical advisers. Dr Prout speaks of a mental imbecility and want of stoicism as one of the effects of the disease. This appears to be the explanation of the assertions that have been made that diabetic patients sometimes pass a quantity of urine exceeding that of the fluids ingested. Niemeyer mentions some observations in which this was at first supposed to be the case, but in which careful watching subsequently proved that the patient had really been drinking a larger quantity of fluid than that to which she had confessed. At Guy's Hospital I have repeatedly seen patients detected in having surreptitiously eaten apples, bread, &c., by the circumstance that the urine has on some one day been found to contain a larger quantity of sugar than had been present before. The advantage gained by daily analysis can therefore hardly be over-estimated.

Now, as I have already stated, the effect of dieting alone, without any medicinal treatment, varies greatly in different cases of diabetes. Sometimes, but very rarely, it cures the disease entirely. Dr Roberts gives a case of this kind. A man, aged thirty-nine, was passing eight pints of urine daily, containing 5680 grains of sugar. Under a restricted diet the mean daily flow of urine went down within a week to 60 ounces; the sugar fell on the third day to 134 grains, at the end of a week to 48 grains, and after a fortnight only a trace of it could be detected. The patient lost all his symptoms, and gained flesh at the rate of three pounds a week. In about eight months the last trace of sugar disappeared. Dr Roberts saw him nearly four years afterwards, and he was then quite well.

But in the great majority of cases the results of dieting are very much less decisive than this. The patient's symptoms may be very much benefited, or altogether removed. The amount of urine which he passes may be greatly diminished, or reduced to the normal quantity. It may even cease to contain sugar, so that for the time all signs of the disease are absent. But if he now attempts to return to his previous way of living, the urine soon again becomes saccharine, and one by one all the symptoms of diabetes reappear.

Indeed, even when the patient thus under dieting loses his symptoms, and the *quantity* of urine which he passes falls to the normal amount, it by no means always ceases to contain sugar. Its specific gravity may be hardly, if at all, lowered; and the percentage of sugar (although not the absolute amount excreted) may be as great as before. Whenever dieting, or, indeed, any other treatment, does good, it appears always to bring the quantity of water down to normal, before affecting to any extent the specific gravity or the proportionate amount of sugar contained in the secretion. And, conversely, whenever dieting readily lowers the specific gravity, it may be assumed that the quantity of urine passed by the patient is not excessive.

Drugs.—But it sometimes happens that a restricted diet fails altogether to control diabetes. There are cases, especially those of very young subjects, in which all treatment appears to be useless. Other patients, again, are at first benefited by dieting; but after a time it ceases to be useful, and the disease appears to advance more slowly when all restrictions as to food are withdrawn. Sometimes the appetite fails entirely as soon as the patient is required to give up amylaceous and saccharine articles of diet.

Now, whenever the limit has been reached of the good that can be effected by dieting alone, the next step should, I think, be the administration of a remedy which has of late been shown to possess great powers of controlling the excretion of sugar in diabetes. I refer to *opium* and to certain of its alkaloids, of which morphia and codeia are the chief. The value of opium in the treatment of diabetes was known to Dr Prout, and

Sir Thomas Watson spoke of it as a treasure. It was also known that patients suffering from this disease could take much larger quantities of the drug than healthy persons. Both the writers, however, to whom I have referred recommended that its dose should as much as possible be kept within moderate limits. But Dr Pavy advocates a very different way of administering opium. He gives it in increasing doses, until he finds either that the disease yields or that the patient is unable to take it. As to the *modus operandi* of opium in diabetes there is difference of opinion. Dr Roberts—who says that, without any restriction as regards diet, daily doses of six to twenty grains always reduce the quantity of urine by about one half—declares that in patients who are dieted it is altogether useless, except to induce sleep and allay irritability. This, however, is inconsistent with Dr Pavy's accurate observations.

But in some cases opium disagrees, either causing narcotic symptoms or (as I have frequently seen) diarrhœa. When the former kind of effects are produced there is often very great advantage in the employment of *codeia*, as was first recommended by Dr Pavy. Half a grain three times a day should be given at first, but the daily quantity has afterwards been gradually raised to as much as thirty grains. Often codeia may be given without any obvious effect but diminution in the amount of sugar excreted. The tongue remains clear and the appetite good. Sometimes, however, codeia causes headache. I have myself repeatedly prescribed this remedy in addition to restricted diet in diabetes, and with most satisfactory results. A young man, aged thirty-two, came to me in March, 1870; he was then so weak that he could hardly walk upstairs, and was passing nine pints of urine daily of sp. gr. 1043. Within nineteen days the quantity of urine was reduced to two and a half pints, it was free from sugar, and of sp. gr. 1018. He already felt stronger, and some time afterwards he returned to active business, and was able to remain at work until eight or nine at night. Four years after, the patient was apparently in perfect health, persevering with both the restricted diet and the codeia.

Alkaline carbonates are said by Dr Pavy and others to be sometimes of service. The waters of Vichy and even of Carlsbad are spoken of highly by foreign writers. Dr Dickinson says that strychnia is valuable as a tonic, and that tincture of perchloride of iron is effectual in removing œdema.

Diabetic patients should be clothed in flannel. Bouchardat maintains that active exercise should be taken.

The intolerable thirst of diabetes may be relieved by dilute phosphoric acid, or solution of bitartrate of potash. Constipation should be treated with Ol. Ricini or Conf. Sennæ, rather than the saline purgatives. The painful sensation of sinking at the epigastrium is said by Dr Roberts to be sooner relieved by two or three grains of assafœtida than in any other way.

It is of interest, as bearing upon the relations of gout to diabetes, that such experienced authors as Prout and Watson agree that mercury is injurious.

BRIGHT'S DISEASE

History—The two more frequent forms—Three others—Common characters—Albuminuria—Casts—Dropsy—Retinitis—Serous and other inflammation—Changes in heart and arteries—Hæmorrhage—Uræmia.

Parenchymatous nephritis—Lardaceous disease—Cirrhosis of kidneys—Consecutive Bright's disease—Cystic degeneration.

General treatment and prognosis in cases of Bright's disease.

In 1827, Richard Bright, of Guy's Hospital, made known the fact that in many cases of dropsy there are well-marked lesions of the kidneys, and that the urine is then albuminous or coagulable by heat. The novelty of the discovery lay in the pathological observations which he had made. That dropsy is often attended with the presence of the serum of blood, or even blood itself in the urine, had been pointed out a short time before by Dr Wells, of St Thomas's Hospital (the author of the 'Essay on Dew'), by Dr Blackall, of Exeter, and in the previous century by Cotugno and by Cruikshank. But, notwithstanding that Wells and Blackall each made autopsies in which they found the kidneys "remarkably hard," they both regarded the presence of disease in these organs as an accident, Blackall's idea being that the reason why serum was excreted in the urine must be that it was in a vitiated state from some cause or other, possibly from having already formed part of the dropsical fluid in one of the serous cavities or elsewhere, and having afterwards been reabsorbed into the blood. On the other hand, scattered descriptions of morbid states of the kidneys accompanied by dropsy are said to occur in the works of Morgagni and some other early writers on morbid anatomy. But to Bright belongs the full credit of having been the first to show that such renal disease is frequent, and to indicate its real relations both to dropsy and to albuminuria, and his name is therefore most justly associated with it throughout the whole world.

A difficulty, however, which has stood in the way of all subsequent writers is that the lesions of the kidneys described by Bright vary very greatly in their characters in different cases. Sometimes the organs are whitish-yellow, large, soft, and smooth; in other instances they may be red, small, hard, and nodulated on the surface. Bright himself admitted three forms of the affection, leaving it an open question whether or not they should be regarded as separate diseases. In 1851 Frerichs, of Berlin, propounded the doctrine that the diverse appearances presented by the kidneys belong to the successive stages of a single morbid process. This view, however, was refuted two years later by Dr Wilks, who showed in a paper in the 'Guy's Hospital Reports' (vol. viii, 2nd series, p. 238) that, under the name of Bright's disease, there are included at least two independent affections differing in their causes, in their mode of onset, their symptoms, and their course—"the large white kidney with considerable dropsy," and "the hard contracted kidney, often destitute of symptoms"—and added a summary of twenty-three and thirty-three cases of each form.

To make clear the argument upon which Dr Wilks mainly relied I must first mention that, although Bright laid so much stress upon dropsy as an effect of the renal affection which he described, he knew from the first that in many cases no dropsy was present. Now, Dr Wilks pointed out that since this symptom is associated in a special manner with the "large white kidney," one ought, if that were an early stage of the "small red kidney," to obtain a definite history of there having been at a former time an attack of dropsy in each case in which the small red kidney was found after death. Frerichs, however, had not brought forward a single instance in which there had been this sequence of events, nor were any furnished by Dr Wilks's own experience. I shall presently have to show that the statement that the large white kidney does not pass on into the small red kidney may perhaps be put too absolutely. But in the main it is undoubtedly correct. Moreover, in the post-mortem room it is not uncommon to find in the bodies of those who have died of various diseases kidneys showing all the stages of the morbid process by which they gradually became shrunken; and this certainly does not involve the occurrence of any enlargement, nor is it attended with any change of colour. As for the other points of distinction pointed out by Dr Wilks, it may be briefly stated that, whereas the large white kidney often results from scarlet fever or from exposure to cold, the small red kidney is not traceable to either of these causes, but in many cases to gout or to lead-poisoning; that whereas the former may occur at any age, the latter is seldom seen in persons under thirty, and is not frequent before the age of fifty; that the former is often sudden in its onset and acute in its course, but that the latter always begins insidiously, and is very chronic in development; and lastly, that although either may be attended with changes in the heart and in the arteries, such changes are far less marked in the former than in the latter kind of Bright's disease, which often appears clinically under the mask of cardiac symptoms, or of apoplexy due to rupture of an artery in the brain.

It would, of course, add immensely to the force of these considerations if it could be shown that different pathological processes were concerned in the production of the two forms of Bright's disease, or even that the same process attacked in the one form one anatomical element of the renal substance, in the other form another. But the pathological process is, in both cases, inflammation; and if not in all cases of exactly the same degree of intensity, it always falls short of producing suppuration. Many years ago Virchow, in his 'Cellular Pathology,' suggested the distinction that in the large white kidney the tubal epithelium, in the small red kidney the interstitial tissue, was mainly affected. Recent observations, however, have shown that this distinction is very far from being absolute. And therefore the two names proposed by Virchow, "parenchymatous nephritis" and "interstitial nephritis," can no longer be regarded as perfectly applicable, though for want of better I shall continue to use them.

From the point of view of strict pathology, therefore, we must admit the fundamental unity of Bright's disease, disregarding the partial histological differences that must be recognised between its different forms. But, as physicians, we are nevertheless quite justified in regarding these as distinct affections when we find that they also differ in their causes, in their symptoms, and in their clinical course. Now, E. Wagner and other recent German writers describe separately "acute Bright's disease," "chronic Bright's disease," and "contracted or granular atrophy of the kidneys." Such a division, however, appears to me to be defective in many respects. It keeps apart cases that should be brought together, and it brings together cases that should be kept apart. As the result of scarlet fever, or of cold, or of pregnancy, a person may be attacked with an acute renal affection; but this, if it does not prove fatal or get well, presently becomes chronic, and may ultimately pass into a stage of contraction and of atrophy. All such cases should be held to belong to a single form of Bright's disease; and as an affection of the tubal epithelium is a more or less conspicuous feature of them, I propose to describe them under Virchow's name of *parenchymatous nephritis.* On the other hand, E. Wagner's "contracted or granular atrophy" includes two sets of cases that seem to me to be essentially distinct; one is the advanced stage of the affection to which I have just been alluding, the other is that of which I have already spoken as the "small red kidney." It is to the latter alone that I shall apply the term *interstitial nephritis,* and for reasons that will hereafter appear, I prefer to call it by another name proposed by Christison, *cirrhosis of the kidney.*

These two forms of Bright's disease, however, are by no means the only ones. That in some cases the primary lesion is a lardaceous change in the arterioles and in the glomeruli of the kidney has long been known, and of late it has become apparent that such cases are far more numerous than was formerly suspected. It is true that the lardaceous affection always tends to become complicated sooner or later with "parenchymatous" and with interstitial lesions. But inasmuch as its causes differ from those of Bright's disease in general, and as there are also differences in its symptoms and in its course, all observers are agreed to keep it apart in their descriptions. I also propose to admit two other forms; one is the affection commonly known as "cystic disease of the kidneys," the other, which has only recently been recognised, is in its origin secondary to lesions of the renal pelvis or of the lower urinary passages, so that it may be termed "consecutive Bright's disease."

It might be thought that the recognition of so many independent forms of Bright's disease would render it advisable to drop the common designation. But, inasmuch as several characters belong alike to them all, it is a great convenience to describe them together. And, moreover, in clinical practice one is not infrequently in doubt as to which form is present. But it can easily be understood that to frame a satisfactory definition of Bright's disease is very difficult if not impossible. It should not, I think, be held to include any renal affection that is merely incidental to some other malady, and subsides if it subsides; such are those that accompany certain forms of heart disease, cholera, erysipelas, diphtheria. Again, it should not include any suppurative lesions of the kidneys. And, lastly, it should not include cases in which a transitory albuminuria occurs without there being reason to suppose that any organic change is present in the kidneys. Taking these reservations into account we may perhaps say that by Bright's disease is meant a diffused non-suppurative affection of the kidneys requiring to be regarded as a substantive disease from a clinical point of view. All forms of it are, or may be, attended with albuminuria, but there seems to be no necessity for giving this as part of its definition.

My description of Bright's disease will fall under the following heads:

1. *Parenchymatous nephritis.*—This, as a rule, is attended with general dropsy. The onset is sometimes sudden, especially when scarlet fever or cold is the cause, but in other cases it may be gradual. At an early stage the kidneys are of natural size, or swollen, and are reddish grey in colour. When the disease is more advanced they become whitish yellow, and sometimes very large. Ultimately they shrink and are granular, but they (perhaps always) retain more or less of the white colour. The urine is at first dense, high coloured, scanty, containing much albumen, tube-casts, and often blood. Later on, it may be pale, watery, with a variable amount of albumen. Retinitis and uræmia are of frequent occurrence. In advanced cases the heart becomes hypertrophied and the systemic arteries are thickened. Recovery is possible when the renal changes do not go too far.

2. *Lardaceous disease of the kidneys.*—This is caused mainly by protracted suppuration or by syphilis. At first the kidneys appear but little altered, except that their tissue gives the characteristic reaction with iodine. But afterwards they commonly become very large, pale-yellow, and waxy looking, this being the result of the supervention of an inflammatory process, identical with that which exists in parenchymatous nephritis. And, finally, they may shrink and become granular. There is no doubt that in many cases that have been set down as examples of parenchymatous nephritis the lardaceous change has been present but has been overlooked. The urine is described as being excessive in quantity, clear, pale, containing much albumen, but rarely blood. But when there is much nephritis it may be scanty, high coloured, and lithatic. General dropsy is exceedingly frequent. But cardio-vascular changes, retinitis, and uræmia are seldom observed.

3. *Cirrhosis of the kidneys, or red granular atrophy.*—This is a slow and insidious affection, of which the chief known causes are gout and lead-poisoning. It is perhaps never seen in early life, but begins to occur in persons between thirty and forty, and beyond this it is frequent at all ages up to seventy. It gradually destroys the renal cortex until this may not be more than a line in thickness; the surface of the organ remains of a red colour, but it becomes very uneven and granular. The urine is abundant, clear, pale, of low specific gravity, it contains only a small quantity of albumen, or there may be none at all for days or even for weeks together. I believe that general dropsy never occurs, unless the affection becomes complicated with parenchymatous nephritis. On the other hand, cardio-vascular changes are very constantly developed and reach an extreme degree. In many cases the patient comes under observation with the symptoms of heart disease, including obstructive dropsy, which affects the dependent parts of the body. Cerebral hæmorrhage is another frequent cause of death.

4. *Consecutive Bright's disease.*—This is seen as the result of such affections as stricture of the urethra, stone in the bladder, compression of the ureters by an abdominal tumour, calculous pyelitis, and scrofulous disease of the kidneys. The kidneys become tough, hard, and whitish; they may be either of good size or shrunken, either smooth on the surface or puckered by cicatrices, or even granular. The general symptoms and the characters of the urine are more or less like those of renal cirrhosis.

5. *Cystic disease of the kidneys.*—The main clinical peculiarity of this affection is that the kidneys are often sufficiently large to be felt as abdominal tumours during life. In most other respects it resembles renal cirrhosis.

But before entering upon a detailed description of these different forms of Bright's disease I must discuss certain symptoms and effects which belong in common to several, if not to all of them. These are albuminuria, tube-casts, dropsy, retinitis, secondary inflammations, cardio-vascular changes, hæmorrhages, and uræmia.

1. *Albuminuria.*—The coagulable substance, or "albumen," which is found in the urine in Bright's disease is, strictly speaking, a mixture of two albuminous compounds, "serum-albumin" and serum-globulin or "paraglobulin," both of which are naturally present in the *liquor sanguinis.* They can be separated by saturating with crystallised sulphate of magnesia; this precipitates the paraglobulin, but leaves the serum-albumin in solution. Estelle found ('Revue Mensuelle,' 1880) that in certain cases of albuminuria in which he investigated the point, sometimes the whole of the so-called albumen, and, as a rule, all but one third of it, was really paraglobulin. The distinction seems, however, to be devoid of practical

importance; both substances react in precisely the same manner to all the usual tests.

The oldest method of detecting albuminuria is by *heat*. The best way of applying it is to pour a good quantity of urine into a test-tube, and then to hold it near the bottom, while one gently warms the upper part of the liquid over a spirit lamp. When the urine is at all strongly acid, any albumen that may be present becomes solidified. Dr Roberts says that at what temperature this occurs depends upon the amount of albumen; if it is very small, no change is perceived until the boiling-point is reached; if it is large, an opaque coagulum forms at a much lower temperature. For urine which is turbid with urates this test is peculiarly applicable. The existence of turbidity from this cause shows that there is sufficient acidity. The first effect of the heat is to redissolve the urates, and to make the fluid transparent; presently the albumen begins to appear and renders it again cloudy. In a column of fluid of some length all three conditions may be seen at the same time; at the bottom, a part which is cold and turbid; above it, one which is warm and clear; still higher, one which is hot and opaque. But if the urine is faintly acid, neutral, or alkaline, the action of heat is less decisive. In alkaline urine it produces no change, even when albumen is present. There is thus a risk of overlooking albuminuria by trusting to this test. It is true that one may avoid the risk by adding a very little acetic acid—just enough to acidify the urine—before beginning to warm it. But this introduces a new source of error; for if the amount of acetic acid be at all excessive, it may itself prevent the heat from throwing down any small quantity of albumen that the urine may contain, unless indeed the urine is also rich in salts, the presence of which is found materially to increase the coagulability of albumen in such circumstances. The safe way, therefore, is, besides the acetic acid, to mix with the urine about one sixth of its bulk of a concentrated solution of common salt, or of magnesian, or sodic sulphate. On heating the liquid after treating it in this manner, the albumen is seen to come down. Salkowski says that the test so applied is not only absolutely conclusive, but unsurpassed by any other in delicacy. If, on the other hand, the urine, instead of being alkaline, is neutral or faintly acid, the application of heat to it while in that condition frequently produces in it an opacity which looks exactly like that due to albumen, but which really consists of a precipitate of phosphate of lime. By adding a little acid, one can of course redissolve the phosphatic precipitate, and so prevent it from being mistaken for albumen. And it may be laid down as a rule of the first importance, that unless the urine has been ascertained to be strongly acid, the presence of albumen in it must never be affirmed as the result of the application of heat alone, and without the subsequent addition of an acid. But there is always the possibility that urine which throws down the phosphate of lime when warmed may also contain albumen. Now, if acetic acid be the acid employed to redissolve the phosphate, it is obvious from what has already been stated that it must be uncertain whether the albumen will show itself or not. If nitric acid be used, coagulation is perhaps sure to occur; but, on the other hand, most observers are agreed that it is unadvisable, with the object of bringing down albumen, to add nitric acid to urine that has just been boiled, because this acid is apt to induce in the hot liquid other changes, the nature of which is not well understood.

These difficulties in the application of heat for the detection of albumen have led me, as a rule, to prefer to it the nitric acid test, except indeed for urine which is already opaque with urates. And even when this is the case, they can commonly be redissolved for the time by slightly warming the test-tube until its contents are raised to about blood-heat. The best way to use nitric acid is to put only a moderate quantity of urine in the tube, to hold it in a very slanting position, and then to let the acid slide gently down its side. The greater density of the acid carries it through the urine, and causes it to accumulate at the bottom, with the urine floating on its surface.* If no albumen is present, the two fluids are separated by a more or less deeply coloured layer, from oxidation of chromogen (p. 350). If there is albumen it forms an opalescent zone, of greater or less thickness, at the line where the acid and the urine meet. When its amount is exceedingly small, the zone may appear only after the interval of a minute or two. It is made more conspicuous by holding up the test-tube against a dark background, as, for instance, the sleeve of one's coat. Employed in this way, the test is very delicate indeed, far more so, I think, than that by heat.

Dr Roberts, however, has shown that the readiness with which albumen is precipitated by nitric acid is to some extent affected by the presence of other dissolved matters. The proof of this is that if two samples of the same albuminous urine be diluted, the one with successive quantities of pure water, the other with the same quantities of healthy urine, the former continues to yield an opaque zone with nitric acid, after the latter has ceased to give any sign of the presence of albumen in it.

I am not aware that this test ever brings down anything but albumen, in such a way as to deceive a practised eye. In urine of high specific gravity urates are sometimes precipitated by it; but, as Dr Roberts remarks, they first appear, not at the line of junction of the two fluids, but at or near the surface of the urine, the turbidity gradually spreading downwards. If there is any doubt it may be removed by gently warming the test-tube, when urates will at once disappear. In the urine of patients who are taking copaiba or cubebs, a resinous substance is excreted, which is precipitated by nitric acid, but not in so well-defined a zone. The application of heat diminishes the opacity from this cause, and the addition of alcohol entirely removes it. I have, however, often seen blunders committed by clinical clerks in the case of patients who are taking the resin of copaiba as a diuretic, and whose urine consequently has been set down as albuminous; and I remember a man under treatment for gonorrhœa, who came out with the copaiba rash, and was supposed to have scarlet fever with his urine already full of albumen!

Many other tests for albumen are known besides the two already given. I may mention acetic or citric acid with *potassium-ferro-cyanide*, and also *potassio-mercuric iodide*. Dr George Johnson has strongly advocated the use of *picric acid*. One advantage which it possesses over nitric acid is that it can be carried about without the risk of its giving off fumes by which other apparatus may be destroyed: another is that it also serves as a test for sugar in the urine, for if picric acid is boiled with potash and diabetic urine, it turns of a deep crimson colour. Dr Oliver, of Harrogate, has brought out a series of papers, saturated with this and other solutions, which are certainly as portable as could be wished. But I am not disposed to give up nitric acid in favour of less familiar tests, inasmuch as it seems to be doubtful whether other substances beside albumen may not sometimes be precipitated by them from the urine. Among these are peptone and propeptone (or hemi-albuminose), a compound intermediate between peptone and albumen. Neither of these two bodies is precipitated by heat; and nitric acid has no effect on peptone, but it at first precipitates propeptone, which, however, is afterwards redissolved by an excess of the acid, with the production of a yellow colour. Tincture of galls has long been known as a precipitant of proteids in urine. Very little seems to be known with regard to the conditions under which peptones or syntonin are found in the urine; but their presence is, at any rate, not an indication of Bright's disease, so that it is important not to confound them with albumen.

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* [A still better plan is to put a little nitric acid into the test-tube first and add the suspected urine to it, letting it trickle down the under side of the tube. The zone takes a minute or more to form if there is only a trace of albumen, but the test is even more delicate than that of heat, much more so than ferrocyanic acid, and more certain than either heat or picric acid.—Ed.]

To determine with absolute accuracy the amount of albumen in the urine takes up a great deal of time; it has to be precipitated from a known bulk of the fluid, washed, dried, and weighed. In clinical practice, however, there is no sufficient object to be gained by this troublesome procedure. Dr William Roberts in 1876 proposed a method which is far easier, and which appears to yield sufficiently satisfactory results. It consists in diluting the urine with water until it almost ceases to give a reaction with nitric acid, the point fixed being that at which the opalescent zone at the junction of the two liquids begins to be visible between thirty and forty-five seconds after the addition of the acid to the urine. To calculate the number of grains of albumen per fluid ounce of urine all that is necessary is to multiply the figure 0·0034 by the number of dilutions with an equal bulk of water that the urine has undergone. A still simpler plan, but one that yields only comparative results, is to take a column of urine of definite depth in a test-tube, and after precipitating all the albumen in it with heat or with nitric acid to let it stand until the coagulum has sunk to the bottom, forming a layer the depth of which can be expressed as a fraction of that of the urine, a half, or a quarter, or one sixth, as the case may be. Vogel, however, found that the space occupied by the same quantity of albumen might vary widely according as it happened to be thrown down in larger or smaller masses, and it was also influenced by the specific gravity of the urine, the range of error from these causes being as much as from 30 to 50 per cent. Even so, however, the method is sufficiently exact for comparison. The actual weight of albumen contained in the most bulky coagulum is but small. Accurate analysis seldom gives more than 5 per cent., even when the urine becomes solid when boiled.

Theory of albuminuria.—In endeavouring to understand why albuminuria should occur, whether in Bright's disease, or under other circumstances, it is necessary to begin by considering how it is that the renal secretion normally contains no albumen. What prevents its escaping from the blood with the water and the other substances which are drained off through the glomeruli? Only one answer to this question seems possible, namely, that it is kept back by the epithelial layer which covers the capillary tufts; and, as Cohnheim remarks, it is interesting to notice that the vessels of the choroid plexuses, which also yield a non-albuminous fluid, are the only ones that have a similar investment. At one time, indeed, a theory was current, according to which albumen was supposed to be present in the transudation from the glomeruli, but to be taken up again and restored to the blood by the epithelial cells of the convoluted tubes; but that notion has been finally exploded by the observations of Posner ('Virch. Arch.,' 1880). But that, when albumen does appear in the urine, it escapes through the glomeruli is rendered probable by the Nussbaum's experiments on frogs ('Arch. f. Phys.,' 1878), in which animals these structures have an arterial supply distinct from that of the renal tubes; he ligatured the glomerular arteries, and found that after this operation egg-albumen, injected into the stomach or into the blood, no longer passed into the urine, as it does when the circulation in the kidneys is undisturbed. Again, Ribbert ('Centralblatt,' 1879), having set up an artificial albuminuria in rabbits with egg-albumen, excised the kidneys, and placed them directly in alcohol, so as to coagulate *in sitû* the albumen in their interior; he then found that the spaces within the Malpighian capsules always contained coagulum as well as the tubes. Such experiments cannot, of course, exclude the possibility that an albuminous fluid may also sometimes transude through the tubal capillaries. But it must be remembered that in that case it would naturally find its way, not into the urine, but rather into the lymph-spaces between the tubes. Consequently, even when albuminuria is dependent upon obstruction to the blood-flow through the systemic veins, and associated with an "obstructive" form of dropsy, it does not seem likely to be due merely to an escape of serum through the tubal capillaries as the result of increased pressure.

What appears to be the most probable cause of albuminuria in general is the occurrence of some nutritive change in the epithelium covering the glomeruli, rendering it no longer capable of resisting the passage of albumen. This view is maintained by Leube and by E. Wagner, as well as by Cohnheim. It is held that whenever a full stream of arterial blood is not kept up through the capillary tufts, their epithelium is liable to be damaged, so that it can no longer fulfil its normal function. The instance which Cohnheim adduces as most obviously supporting such an opinion is that of the albuminuria which follows the suppression of urine during an attack of cholera. This, he maintains, is precisely analogous to the albuminuria which can be experimentally produced by temporary obstruction of the circulation through the renal artery, and which lasts for hours or even days after the obstruction is removed. But another cause of deficiency of blood-supply to the glomeruli may be an impeded outflow through the veins of the kidneys. As a matter of fact, Ribbert has shown that after arrest of the circulation through the renal artery the cells of the glomerular epithelium become obviously swollen and altered in appearance. But, in many other cases it is a mere assumption that any change occurs in them, nothing having as yet been detected with the microscope.

According to this conception of the pathology of albuminuria, it has no essential relation to the state of the blood-pressure on the vessels of the kidneys. Until lately, the prevalent doctrine has been that nothing favours the escape of albumen so much as an increase of blood-pressure. This was the conclusion to which Stockvis arrived at as the result of his elaborate investigations, and it was adopted by Bartels. According to Cohnheim, however, it is based upon no evidence whatever, whether experimental or pathological. The occurrence of albuminuria as the result of venous obstruction certainly lends no support to it, because the pressure in the glomeruli is probably thus diminished rather than excessive, in consequence of the enfeeblement of the heart's action which arises at an early period in such cases: and Runeberg actually maintains that abuminuria is always dependent upon a *deficiency* of blood-pressure.

There does not appear to be any more foundation for another theory of albuminuria, according to which it depends upon a change in the albuminous elements of the liquor sanguinis, enabling them to pass with undue facility through the walls of the glomeruli. Stockvis ingeniously disproved this notion by the direct experiment of injecting albuminous urine from patients with Bright's disease into the veins of animals, when he found that the albumen did not, as a matter of fact, escape from their kidneys. The same observer failed altogether to obtain experimental corroboration of the idea, formerly common, that hydræmia may be a direct cause of albuminuria.

"*Physiological*" *albuminuria.*—If now we pass on to discuss the conditions under which albuminuria occurs, we find, in the first place, that it is seen in many persons who are, so far as can be ascertained, in good health, and whose kidneys appear to be perfectly sound. It is only within the last few years that this fact has been clearly ascertained. Leube tested ('Virch. Arch.,' 1878), the urine of 119 soldiers, and found albumen in the urine passed in the morning by five of them, and in that passed at midday after a march by no fewer than nineteen ; the urine passed in the evening was never albuminous. Fürbringer ('Ztschft. f. klin. Med.,') tested the urine of sixty-one children, and detected albumen in seven cases, always in the latter part of the forenoon. In other instances the presence of albumen in the urine of healthy persons has been traced to some definite cause. Thus Dr George Johnson has recorded ('Clin. Soc. Trans.,' vol. vii) several cases in which it was temporarily produced by cold bathing; the same observer ('Brit. Med. Jour.,' 1879, ii) alludes to other cases in which it followed active walking exercise, an instance of which has also been related to me by a medical man of my acquaintance, as having occurred in his own person ; Fürbringer relates a case in which it was more than once brought on by distress of mind. Again, Dr Moxon has related in the 'Guy's Hospital Reports' for 1878, several cases (altogether nineteen) in which albumen was from time to time discoverable in the urine of boys and young men who were generally anæmic, listless, and languid ; with all in whom he was able to trace the further progress of the affection, it sooner or later passed off, usually in the course of a few months. Dr Dukes, of Rugby, shortly afterwards stated that he had seen ten cases in boys of thirteen to seventeen, in whom albuminuria had occurred as the result of cold, exertion, or excitement, but subsided when they were kept in bed and on a milk diet. In some of Dr Moxon's cases, "albuminuria of adolescents" (as he terms it) was associated with oxalate of lime. E. Wagner speaks of having seen similar cases in anæmic and weakly girls; and so has Sir William Gull.

The view which is taken by recent German writers with regard to what they call "physiological albuminuria". is that it depends upon a congenital deficiency in the power of the glomerular epithelium to resist the passage of albumen through it. It may perhaps be urged in support of such a theory that there are two pairs of brothers among the seven cases of which Dr Moxon gives details; and Leube also mentions having met with the affection in two brothers. But if it were correct one might fairly expect that the urine, if not constantly albuminous, should at least become so under the operation of definite disturbing causes. Dr Moxon states that two of his patients had been previously under his observation, and that the urine was then in each case invariably free from albumen.

But the most important question of all is whether in such cases the occurrence of albuminuria indicates any tendency to the development of organic renal disease. Ought a young man, in whom this affection is discovered, to be regarded as eligible for life insurance at the ordinary rate? Leube and Fürbringer would doubtless answer this question in the affirmative, and so, I think, would Moxon, if it were clearly ascertained that the urine contained albumen only occasionally, and that in the forenoon. I may note that both Fürbringer and Moxon detected a few hyaline casts in more than one instance, so that casts cannot be taken as affording conclusive evidence of serious mischief in the kidneys. And it seems to be clear that the cases recorded by the different writers whom I have cited are far too numerous to be set down as examples of latent Bright's disease. Had they all been of that nature we may be sure that some of them would have revealed their real character while they were still under observation. On the other hand, Dr Johnson has expressed ('Brit. Med. Jour.,' 1879, ii) a decided opinion that temporary albuminuria, even when traceable to food, or exercise, or exposure to cold, will, if neglected, almost certainly sooner or later lead to a persistent albuminuria, and this ultimately to fatal disease of the kidneys. Of the fact that it is possible for albuminuria to be the only indication of ill-health and yet for the kidneys to be undergoing grave structural changes there is no doubt whatever. Dr Johnson mentions the case of a medical man, actively engaged in a large practice until shortly before his death from uræmia at forty-five years of age, whose urine had been albuminous from the time when he had been a student, and probably earlier still, since he had scarlatinal dropsy when fifteen years old. Clearly, therefore, an insurance office which ignored the presence of albuminuria in applicants who appeared otherwise well would sometimes lose thereby. But I am inclined to think that in many cases in young men, if the disorder were ascertained to be only occasional, and if there were no history of a former attack of nephritis, the risk might be fairly accepted with a moderate addition to the premium. Probably some at least of the cases in which the urine is found to be temporarily albuminous after sea-bathing or exposure to cold in other ways are related rather to the paroxysmal hæmoglobinuria which, as we have seen, appears to have no tendency to pass on to the development of organic renal disease. But this does not apply to the first of Dr. Johnson's cases, in which the albuminuria persisted for nineteen days after bathing. The only insurance company that, so far as I know, has made any attempt to ascertain the subsequent state of health of persons whose lives had been declined on account of albuminuria is the United States Company in the City of New York. Among those who made applications to that office in the three years 1878—1880 there were sixty-nine (or from 10 to 12 per cent. in each year) whose urine was found to be albuminous. Before the end of 1880 four of these persons died, and it is stated by Dr Munn that the "general appearance of the majority of the others who had been under observation for more than a year was gradually deteriorating." It is to be noted, however, that few of them were under the age of thirty, and that the albumen was often present in considerable quantity. Consequently, although the results of this investigation show that an insurance company runs a great risk if it neglects to have the urine of applicants tested, they can hardly be said to throw much light upon the question of the occurrence of a physiological albuminuria in young subjects. On the other hand, it appears to be a significant fact that Mr Eales, of Birmingham, found retinal changes in five out of fourteen cases of supposed temporary albuminuria in persons between the ages of eleven and twenty-eight ('Birmingham Medical Review,' 1880).

Albuminuria in disease.—Albumen occurs in the urine without there being any marked or permanent lesions of the kidneys, under various morbid conditions, which may be briefly enumerated under the following heads:

1. Obstruction of the general venous circulation as the result of heart disease, emphysema of the lungs, and other like affections.

2. Obstruction of the renal veins, independently of any disease affecting the whole circulation, as in a case recorded by Bartels of obliteration of the inferior vena cava above the mouths of those veins.

3. Deficient blood-supply to the kidneys through the renal arteries, as in cholera. The explanation of the albuminuria from this cause has been fully discussed already at p. 295 of the first volume.

4. Obstruction of the ureters, the albuminuria of course appearing only after the obstruction is removed. This cause has been established by experiments on animals, and a case in point is given by Bartels in which the obstruction was produced by a calculus.

5. Various febrile maladies, especially acute pneumonia, but also typhus, enteric fever, cerebro-spinal meningitis, erysipelas, ague, pyæmia. In scarlet fever and diphtheria too during the pyrexial stage there may occur albuminuria, which probably ought to be distinguished from that which appears at a later period and is dependent upon nephritis. Cloudy swelling of the renal epithelium is constantly found in the bodies of those who have died of febrile maladies, but it is not held that this has anything to do with the albuminuria, which is far less frequent.

6. Certain affections of the nervous centres, especially cerebral hæmorrhages, concussion of the brain, epilepsy, tetanus, delirium tremens.

7. Affections attended with some abdominal pain and collapse. This cause of albuminuria has been especially pointed out by Fischl ('Deutsch. Arch.,' 1881).

8. Various chronic diseases, including leuchæmia, diabetes, exophthalmic goitre, and also (it is said) anæmia and tuberculosis.

9. Poisoning by different substances.

If now we ask what explanation can be given of the origin of albuminuria under these several conditions, the answer is, I think, unsatisfactory, except in the case of cholera. Taking, for example, febrile albuminuria, we are quite unable to say whether the invisible physical change in the epithelium of the glomeruli, to which (as we believe) must be attributed the escape of albumen through them, is due to the heat of the blood itself, or to the action of the heat upon the renal nerves, or to chemical changes in the blood, or to disturbance of the circulation through the kidneys as the result of diminished arterial pressure. When albuminuria follows an epileptic fit, or an attack of cerebral hæmorrhage, it may be doubted whether it is not a secondary result of venous obstruction from impeded respiration. But the albuminuria dependent upon venous obstruction is itself susceptible of various interpretations. I have already alluded to one view, according to which it depends upon a deficient supply of arterial blood to the glomeruli, interfering with the due nutrition of their epithelium. But another idea has been that distension of the veins at the line of junction of the renal cortex with the pyramids may compress the straight tubes, and so interfere with the flow of urine, and exert pressure backwards in the Malpighian capsules upon the outer surface of the glomeruli. Commonly, it has been thought that when albuminuria follows plugging of the ureter, the distended renal tubes press upon the veins; and thus that this cause of albumen in the urine may after all be included under the head of "venous obstruction." Evidently, hypothesis has here at present an unlimited range.

Albuminuria in renal disease.—Turning now to the albuminuria which accompanies disease of the kidneys, we find that in one very important particular it differs from that which occurs under all other circumstances, namely, in the much larger amount of the albumen, both absolutely and in proportion to the urine. In Bright's disease, for example, the urine may contain 5 per cent. of albumen, a quantity more than half as great as that in normal blood-serum. It is true that the proportion is generally much smaller; but then we must remember that the urine as we obtain it is, after all, a mixture of the fluids poured out by an almost infinite number of glomeruli and renal tubes, which, if they are not all diseased to a like extent, may yield secretions of very different quality. So again, in cases in which renal infarctus, or localised new growths, are surrounded by zones of hyperæmic and inflamed kidney-tissue, any albumen that may be contained in the secretion from these parts is necessarily distributed over the very much larger quantity of fluid poured out of the rest of the cortex, which may be quite healthy. It is undoubtedly to changes in the glomeruli that we must mainly attribute the albuminuria of Bright's disease, though, perhaps, the renal tubes may also furnish a share of it; whether this is more likely to be the case when their basement membrane has been exposed as the result of exfoliation of the epithelium must at present, I think, be regarded as doubtful.

2. *Tube-casts.*—The discovery of these bodies—"urine-cylinders," as the Germans call them—in the urine is generally associated with the name of Henle, who described them in 1844; but they had not escaped the notice of some previous observers. There are several different varieties of them.

The most frequent and typical are those known as "hyaline" casts. These are delicate, transparent, colourless, with defined outlines, but so little refractile that they are not always recognised under the microscope in the fluid in which they float, unless stained by carmine or iodine or aniline dyes. They vary in breadth from 0·01 to 0·05 mm.; their length may be only a few times greater than their breadth, or may reach 1 mm. or even 2 mm. They may be either straight, or curved, or bent. Interspersed through their homogeneous substance, or adhering to their surface, there are sometimes red blood-discs, leucocytes, more or less altered epithelial cells, fatty granules or drops of various sizes, granules of urate of soda, or crystals of oxalate of lime. I remember one case of Bright's disease in which for some weeks all the casts contained numerous red blood-discs, to the exclusion of all other formed elements. In other instances, tube-casts look as if they consisted almost entirely of epithelial cells, packed so close together that little or none of the hyaline material can be seen; sometimes these are distinguished by the name of "epithelial casts." Or they may appear to be made up of a reddish-brown substance, which is believed to be derived from altered blood-discs, and they are then called "hæmatoidin-casts." The fat-granules or fat-drops are probably always derived from disintegrating epithelium; casts in which they are abundant are known as "fatty casts," and may look quite opaque and black by transmitted light. The term "granular casts" appears to be used somewhat differently by different writers. E. Wagner describes them as "opaque like ground-glass," as "not seldom appearing as if eroded or breaking down at their edges," and as "sometimes presenting numerous indentations, or looking as though they were made up of a number of square pieces fused together." Lastly, casts are sometimes, though very rarely, "waxy" or "lardaceous;" they then assume a red-brown colour with iodine; they are highly refractile, and they show far more resistance to reagents than the common hyaline casts. Bartels is disposed to admit the possibility of their acquiring the lardaceous character as the result of age, when they are long retained in the renal tubes. Some writers say that their occurrence is not peculiar to cases in which the kidneys are themselves lardaceous, and that they may be found in other chronic forms of Bright's disease.

Under the name of "cylindroids,"—for which "false casts" would perhaps be the best English equivalent,—some recent German observers have described certain flat, riband-like bodies, which are found in the urine of patients with scarlet fever, and also in cases of cholera and of recurrent fever. They are pale, homogeneous, colourless, but reach a much greater length than ordinary hyaline casts. E. Wagner says that their nature is still unknown. Some think that they are of a mucous character, and that the urine does not contain them until after it has escaped from the pyramids. Others are of opinion that they are found in the renal tubes. It does not appear clear that there are any transitional forms between "false" and "true" casts, though they may be found together in the same specimen of urine.

After death, casts may be seen in the kidneys in every part of their substance, from the convoluted tubes near the glomeruli down to the wide collecting tubes in the pyramids. They are most abundant in the looped tubes. E. Wagner thinks that the reason is that they are slow in passing through these narrow canals. Some writers have doubted whether casts from the convoluted tubes are capable of traversing the looped tubes so as to be discharged with the urine. But the better opinion seems to be that they are so elastic and flexible that this is not impossible. And it may be that materials that originally solidified in the highest tubes close to the glomeruli may afterwards be, so to speak, re-cast, taking the form of tubes lower down.

It is stated, both by E. Wagner and by Bartels, that very wide, waxy, or granular casts are found chiefly in cases in which the urine is very scanty, and especially in the chronic forms of Bright's disease, within a few days of the fatal termination; and Bartels observes that such casts, moulded in the collecting tubes of the pyramids, can be retained there long enough to undergo secondary changes only when the secreting activity of the kidney is at a very low point.

The chemical nature of the hyaline material which appears to be the basis of all recently formed tube-casts has been especially studied by Rovida, who arrived at the conclusion that it is not identical with either fibrin or albumen, so that it can only be described as an *albuminoid* substance. There have been various opinions with regard to the mode of origin of casts. Some observers have supposed them to be produced by a process of secretion on the part of the epithelial cells of the tubes. Bartels upholds this view on the ground that spheroidal masses of plasma can often be seen protruding from the cells into the lumen of a tube. E. Wagner, however, says that such appearances may be observed even in healthy kidneys. Another theory has been that they arise by the fusion together of altered epithelial cells. According to Weigert ('Volkmann's Sammlung,' 162-3) this is obviously the case in animals in whom nephritis is set up by the injection of chromate of potass under the skin. And in Bright's disease such an origin seems probable in the case of certain casts which have indented margins or look as if they were made up of an agglomeration of angular pieces. Some writers think, too, that casts which turn reddish-brown with iodine are formed out of epithelial cells that have first become lardaceous. But for the ordinary hyaline casts by far the most probable view is that they result from the coagulation of fibrinogen exuded from the glomeruli, under the influence of some substance yielded by disintegration of leucocytes. The fact that their reactions are not identical with those of fibrin may perhaps be explained by the supposition that they undergo some further chemical change under the influence of the acid urine which bathes their surface. The very short time which sometimes passes between the commencement of a morbid change in the kidney and the appearance of casts in the urine affords a strong argument in favour of the view that they arise by such a process of coagulation. Bartels, for instance, states that in a patient who underwent the operation of transfusion with lamb's blood, and whose urine up to that time was normal, urine passed two hours afterwards contained not only albumen, but also hyaline casts. In another case, that of a man who fell from a height upon his sacrum, urine voided five hours later showed hyaline, as well as blood-casts. There is indeed a very clear relation between albuminuria and the presence of tube-casts. In some cases, however, they appear in the urine a few hours, or even a day or two, before albumen is discoverable in it; and sometimes, perhaps, their presence continues without albuminuria developing itself. In the urine of jaundiced patients casts of a greenish-yellow colour are often found. Dr Finlayson says that, as a rule, in such cases, no albumen is present. Dr Roberts alludes to cases of venous obstruction from heart disease or emphysema as being also accompanied with renal tube-casts, although there is no discoverable albuminuria, but he adds that in most of these cases the urine is of high specific gravity, so that the absence of a trace of albumen in it can hardly be affirmed with certainty. Of course nothing is proved by the fact that in cases of acute Bright's disease casts sometimes continue to be passed after albuminuria has ceased, because they may have been retained in the renal cortex for a considerable time after their formation. As a rule, the abundance of casts in a case of Bright's disease is fairly proportionate to the amount of albumen in the urine; but to this there are many exceptions, and in the same patient the number of casts may vary greatly from day to day.

It is generally said that tube-casts possess great clinical importance from the fact that they prove the substance of the kidneys to be diseased. This is undoubtedly so far true, that in cases in which the urine contains pus or blood, which may have been derived from the renal pelvis or the lower urinary passages, the discovery of casts may be the sole indication that the renal cortex is affected. But in such instances if none can be found it proves little or nothing, for their recognition is very difficult when leucocytes or red blood-discs are present in numbers. On the other hand, if there is merely albuminuria, the presence of casts must not be supposed to be evidence of the existence of "Bright's disease" rather than of those slight or temporary changes in the glomeruli which occur in association with pyrexia, or as the result of obstruction to the venous circulation. E. Wagner also mentions that he has sometimes found casts in cases of tubercular disease of the kidneys.

3. *Dropsy.*—In speaking of the discovery by Bright of the disease that has since borne his name I mentioned that the cases in which he learned to look for it were mainly cases of dropsy, and this has always been recognised as one of its chief symptoms.

As a matter of fact, indeed, there occurs in Bright's disease two kinds of dropsy. One is identical in its characters with that seen in heart disease or in emphysema of the lungs, and depends upon obstruction of the systemic veins. When it appears in Bright's disease, it is only an indirect effect of the primary malady, its immediate cause being failure of the heart to maintain the needful activity of the circulation. It is always more marked in the dependent parts of the body than elsewhere, and especially in the lower limbs, and it is associated with dyspnœa, with orthopnœa, and often with lividity. It occurs only in the chronic forms of Bright's disease, and especially when the kidneys are "red and granular" or cirrhotic.

Widely different from this is the kind of dropsy which, although perhaps not absolutely more frequent, has been always justly associated with Bright's disease as being almost, if not quite, peculiar to it. This kind of dropsy often begins in the face, about the eyelids, even before it affects the ankles. I do not say that its distribution is altogether independent of the influence of gravitation, for one may often notice that whereas the face is swollen when the patient rises in the morning, the œdema subsides towards the latter part of the day. But at any rate it is not limited to the dependent regions of the body like the other kind, and it is not accompanied by any signs of cardiac disorder, such as dyspnœa or lividity. Indeed, the whole of the body and the limbs often swell at the same time, and acquire a peculiar white waxy appearance, which is very characteristic. The occurrence of such a general dropsy is frequently the earliest symptom of Bright's disease, and first draws the patient's attention to the fact that he is unwell. Generally, however, the urine is found, if tested, to be already albuminous; and after scarlet fever, when the supervention of dropsy can be anticipated as likely to happen, albuminuria may be known to be present for several days before any œdema can be detected. On the other hand, it sometimes happens that the dropsy precedes the albuminuria by a day or two.

Cases are now and then met with in which there is dropsy of precisely the same character as that which is so constantly associated with Bright's disease, but in which no albumen can at any time be found in the urine. Such cases are sometimes dignified by the name of "*essential dropsy,*" but I think that one may fairly doubt whether the kidneys are healthy, although no clinical evidence to the contrary can be obtained. What, however, is more frequent is for a patient to come under observation with general dropsy that has already lasted several days, or even weeks, and for his urine to yield no coagulum, either then or at any subsequent period, while the dropsy more or less rapidly subsides. In these cases, of which I have seen several, nothing is more likely than that albuminuria was really present at first, for it is well known that in the more transitory forms of Bright's disease the urine often becomes normal before the dropsy disappears.*

I shall leave the clinical characters of the general dropsy of Bright's disease to be described in detail when I am discussing that form of the disease, parenchymatous nephritis, in which it chiefly occurs. What I mainly wish to discuss in this place is the theory of its origin. Now, the most obvious suggestion with regard to it is that it depends upon an altered state of the blood, the result of the perverted action of the kidneys. Both Bostock, who made analyses of the blood for Bright himself, and many later observers, have found that the density of the serum is greatly reduced, being not more than 1020, or even 1013, instead of the normal density of 1030. It is natural to refer this physical change in the blood partly to the deficient excretion of water, partly to the abnormal escape of albumen, through the glomeruli of the kidneys. Some writers have laid special stress upon the loss of albumen, and the resulting "hypalbuminotic" state of the blood, as being the main cause of renal dropsy. But Cohnheim points out that the amount of albumen which is excreted by the kidneys is after all inconsiderable. In most cases the percentage of albumen in the urine does not exceed 2 per cent.; in exceptional instances it may reach 4 or 5 per cent., but then the quantity of urine passed in the twenty-four hours is always much diminished, so that after all the total daily loss of albumen cannot be calculated at more than from eight to ten or twelve grammes (two to three drachms). It is obvious that, unless the assimilation of food is greatly interfered with, such an amount of albumen can be very easily replaced. And as a matter of fact, quite as large quantities of albumen are lost, without any dropsy resulting, by patients with large granulating wounds, and by those who have chyluria; and far larger quantities by women during lactation. Consequently, it has been urged by Bartels and others that the really important factor in the production of renal dropsy is the deficient excretion of water by the kidneys. Rehder is cited by Bartels as having made a very elaborate series of investigations, in several cases of Bright's disease, as to the relation between the amount of water drunk (that contained in the solid food being, however, left out of consideration) and that discharged in the urine from day to day. And in one case particularly he found that during periods when the dropsy was on the increase the water excreted was not more than from 29 to 49 per cent. of that which was ingested, whereas during periods when the dropsy was decreasing, the ratio was from 72·5 to 100·5 per cent. But, as Cohnheim remarks, such observations, after all, warrant no conclusion as to the nature of the connection between scantiness of the urine and dropsy. One has just as much right to suppose that the variations in the dropsy caused those in the activity of the kidneys, as to take the common view. The effect upon the blood of a deficient excretion of water by the kidneys, supposing it not to be corrected either by a deficient ingestion of water, or by an increased loss of water through some other channel, must obviously be to increase the whole bulk of the circulating fluid, while diminishing the percentage of solids in it. Cohnheim expresses this by saying that the resulting state of the blood must be not a mere "hydræmia," but a "hydræmic plethora." Now, he and Lichtheim

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* [On this point see some cases in children recorded by Dr Duckworth in the 'St Barth. Hosp. Rep.' I have seen the same twice in adults, one a young man in hospital, the other a married woman; both cases were watched throughout.—Ed.]

('Virchow's Arch.,' lxix) made a series of experiments upon dogs, in which they found that the injection of enormous quantities of a half per cent. solution of salt into the blood produced not the slightest anasarca, even when the renal arteries were ligatured, so as to cut off the escape of the fluids through the kidneys. So far, therefore, as experiment can settle the question, it appears that a "hydræmic plethora" is incapable of causing the dropsy of Bright's disease. But, in fact, there is no evidence whatever that such a condition of the blood occurs in this disease, or can arise as the result of impairment of the renal functions. Unfortunately, nothing is positively known as to the amount of water which escapes from the lungs or from the skin, though it must be admitted that the dry harsh state of the cutaneous surface in many cases of Bright's disease, and the difficulty with which visible sweating can be induced, render it unlikely that the skin takes up any part of the renal function. But there can be little doubt that in one way or another the inactivity of the kidneys is compensated for, and that the volume of the blood remains unaltered or nearly so. And further, there is abundant clinical proof that even complete arrest of the secretion of urine, arising under other circumstances, causes no dropsy. Not to mention the anuria of hysterical women, there are the cases of "obstructive suppression" resulting from plugging of the ureter of a single kidney in man, the other kidney having been previously destroyed by disease (*cf.* p. 388). And in animals, again, ligature of the ureters is equally incapable of producing such an effect. Lastly, in many cases of scarlet fever dropsy sets in before there is evidence of impairment of the renal functions, and certainly long before there has been time for the development of any great change in the density or in the volume of the blood as the result of such impairment.

Such considerations render it clear that some further explanation is needed of the occurrence of general dropsy in Bright's disease; and this is sought for by Cohnheim in a change which he supposes to take place in the walls of the capillaries, rendering them more readily permeable by fluids than they are in normal circumstances. In most cases the "hypalbuminotic" state of the blood might well be imagined to produce such a change. But this view is inconsistent with the fact that in some instances the dropsy sets in before there can have been time for the blood to become "hypalbuminotic" (or subalbuminous). Cohnheim therefore falls back upon the ingenious suggestion that the vessels of the skin and of the subcutaneous tissue become altered by the same cause which sets up the renal affection. He points out that whereas dropsy accompanies the nephritis that follows scarlet fever or exposure to cold, no such result is observed when a like nephritis arises in the course of diphtheria or of relapsing fever, in which diseases the skin remains intact. Obviously, the explanation is valid only so far as anasarca is concerned. And Cohnheim accordingly insists that dropsy of the serous cavities and of mucous membranes does not occur in most cases of Bright's disease, at least in an early stage, when failure in the heart's action cannot be supposed to play any part in their production. But my impression would certainly rather be in accordance with the experience of E. Wagner, that in autopsies upon some most acute cases, as, for instance, after scarlet fever, one generally finds some fluid effused into deeper parts of the body, though not, perhaps, in very large quantity.

It is evident that, as Cohnheim himself points out, the hypothesis of a change in the capillary walls, as the immediate and fundamental cause of renal dropsy, brings the affection somewhat closer than before to the inflammatory forms of œdema. But in one respect there is an important difference, namely, as regards the composition of the effused liquid. This, in Bright's disease, has always an extremely low specific gravity, and contains but a very small quantity of albumen; in fact, it exactly resembles in these points the liquid that is poured out in the "mechanical" dropsy of heart disease, or of pulmonary emphysema. C. Schmidt found in one case that the dropsical fluid from the subcutaneous tissue contained 0·36 per cent. of albumen, that from the meninges 0·6—0·8 per cent., that from the peritoneum 1·13 per cent., that from the pleura 2·85 per cent. Bartels examined fluids taken directly after death from different parts of the body of a person who died of advanced dropsy, and found that the specific gravity of the blood-serum being 1015·60, that of the pericardial fluid was 1009·7, that of the peritoneal fluid 1009·6, that of the anasarcous fluid 1007·65; in each of the dropsical fluids the main part of the solid constituents was made up of inorganic salts. Urea, in the proportion of about 0·3 per cent., was detected in anasarcous fluid, and also in ascitic fluid by Edlessen; in pericardial fluid he once found as much as 1 per cent. of urea.

4. *Albuminuric retinitis.*—One of the most characteristic indications of Bright's disease is, in some cases, the presence of changes in the retinæ. These are said to have been first noticed post mortem by Türck, in 1850; but the discovery of their importance in relation to kidney disease is assigned to Heymann, in 1856. They occur only in cases which are already chronic: in advanced stages of the renal affection after scarlet fever or during pregnancy, and of the insidious form of parenchymatous nephritis; when the kidneys are cirrhotic; very seldom in cases of lardaceous disease, and probably only when it has long been associated with other structural lesions. Sometimes, however, the recognition of changes in the retinæ by means of the ophthalmoscope is the first thing which suggests that the patient is out of health. Their frequency is believed by Dr Gowers to correspond fairly with the statement of Eales, who found them in 28 out of 100 cases of chronic Bright's disease, or in about 1 of 3½ cases ('Birm. Med. Rev.,' 1880). They vary in character in different instances, but they are commonly all included under the name of "albuminuric retinitis," although this is not always quite appropriate.

The most common form of this affection is, in fact, one which seems to be merely degenerative. It consists in the formation of *whitish spots*, sometimes close to the optic disc, sometimes elsewhere; near the macula lutea they often appear as fan-like streaks. They may be round dots, so minute as to be only visible by the direct method of examination; or they may be large irregular patches, which equal the disc in size, and which may coalesce into large areas surrounding the disc. A less intense diffuse opacity is often present over more or less of the retina. Associated with the white spots, or occurring independently of them, *hæmorrhages* are very frequently observed. These lie, for the most part, in the nerve-fibre layer of the retina; and they therefore are often "flame-shaped" (to use Dr Gowers' expression), following the linear course of the fibres. They may also run by the side of and parallel to the vessels. When they are large they may be irregular in shape, or may penetrate into the deeper layers of the retinæ. In some cases, again, *optic neuritis* may be the most conspicuous retinal change. And if no white spots are discoverable, the appearance may be identical with that which is so commonly produced by intracranial disease, and it may ultimately run on to atrophy in exactly the same manner. Lastly, there may be a general *œdema* of the retina, with complete obscuration of the disc. The arteries are narrow and to a great extent concealed; the veins distended and tortuous. There are always many hæmorrhages, forming large streaks in the course of the nerve-fibres. White spots are commonly numerous, large, rounded, and soft-edged. Dr Gowers, from whom the above description is taken, says that this form of the albuminuric affection is confined to cases of severe and rapidly fatal Bright's disease, so that there is rarely time for it to subside or to pass into an atrophic stage.

Anatomically the white spots depend upon a degenerative change in the nerve-fibres, which become greatly thickened, varicose, and filled with fat-globules; "compound granule-masses too appear in large numbers." The vertical fibres of Müller become swollen and fatty. The fanlike distribution of the spots near the macula lutea is said to depend upon the peculiar arrangement of these fibres in that position; they radiate from the *fovea centralis,* and their direction is somewhat oblique. The diffuse opacity is due to œdema, which gives rise, in hardened preparations, to the appearance of cavities, separating the retinal elements from one another. Dr Gowers' drawings of microscopical sections show how greatly some of the layers (including even the layer of rods and cones) may be thickened where there is a white spot.

With regard to the *pathology* of these retinal changes but little is yet understood. Traube was disposed to insist on the fact that they scarcely ever occur except in cases in which cardiac hypertrophy is already present, as showing that the heart affection was mainly concerned in producing them; and Cohnheim still upholds the same opinion. But probably the material fact is rather that albuminuric retinitis requires a considerable time for its development, so that before it appears the heart is almost certain to become enlarged. Dr Gowers draws attention to the small size of the retinal arteries, which in some cases of chronic Bright's disease are seen with the ophthalmoscope to be not more than one half or even one third of the size of the veins; but he regards this as the result of a vital contraction of their coats, and not of any organic changes in them. My colleague, Dr Brailey, however, has shown that these vessels become affected with an *endarteritis obliterans,* exactly like that which we shall presently find to occur in the arterioles of the kidneys and of other parts of the body. Dr Gowers describes and figures irregular dilatations of the capillaries, with increase of the nuclei in their walls; he thinks it probable that such dilatations often lead to hæmorrhages. As for the optic neuritis, which we have seen to be sometimes the main ophthalmoscopic change, he has observed this especially when there have been conspicuous symptoms of cerebral disturbance, such as intense headache, delirium, and convulsions. He is therefore disposed to regard them as the cause of its predominance over the other retinal lesions in the cases in question.

Albuminuric retinitis in its less intense forms may be altogether unaccompanied with subjective symptoms. Or there may be more or less marked amblyopia which may cause the patient to seek professional advice. Voelckers speaks of cases in which transitory darkening of the field of vision occurs from time to time during excitement or on exertion. Even in the most severe forms of albuminuric retinitis it rarely happens that central vision is lost or that complete blindness follows. It is to be observed, too, that the occurrence of defective sight in a case of Bright's disease may depend upon very different causes. E. Wagner remarks that, altogether apart from the presence of retinal changes, it is not uncommon for hypermetropic patients whose general health is much affected to complain of impairment of vision which is really due to failure of accommodation. And I shall presently have to speak of amaurosis as an effect of uræmia.

It must not be supposed that any retinal changes are in themselves absolutely conclusive as to the existence of Bright's disease. Dr Gowers relates a case in which a chlorotic girl became affected with what seemed to be an idiopathic neuro-retinitis, which ultimately left appearances undistinguishable from those of albuminuric retinitis. And sometimes there are difficulties in the diagnosis of the retinal effects of renal from those of cerebral disease. When the white spots are very small, and limited to the region of the macula lutea, they require careful looking for, and dilatation of the pupil by atropine is often necessary to enable one to make sure of not missing them.

As a rule, when albuminuric retinitis has once developed itself, it persists until the patient's death. The exact appearances, however, vary from time to time; hæmorrhages may disappear and fresh ones may form; even the white spots may subside, though Dr Gowers says that this is very rarely the case with those that surround the macula lutea. It is in the Bright's disease associated with pregnancy that there is most ground for hoping for a permanent recovery from the retinal affection. The free use of purgatives is believed to favour its subsidence and to diminish the tendency to recurrence.

5. *Secondary inflammations.*—Among the most serious effects of Bright's disease as being frequently the direct cause of death, must be mentioned the occurrence of inflammation in one or more of the serous cavities, or in the lungs. Of the different serous membranes, the pleura, the pericardium, and the peritoneum are most apt to be attacked; meningitis is very rare, and perhaps, even where it does seem to be a result of renal mischief, some other well-accredited mode of origin for it ought to be found, if it were carefully looked for. The general tendency of the serous inflammations due to Bright's disease is to become suppurative; this is particularly well marked in the case of the peritoneum; but, on the other hand, I do not remember to have ever seen a purulent exudation in the pericardium, even in the meshes of lymph. In the several forms of Bright's disease there are some differences as regards the liability of one rather than another serous membrane to become affected. Why such secondary inflammations should arise is not very clear. They are commonly attributed to the impure state of the blood which results from defective excretion on the part of the kidneys.

6. *Cardio-vascular changes, hypertrophy of the heart, and thickening of the arteries.*—From the time of Bright himself it has been known that hypertrophy of the heart is frequently associated with kidney disease; and since the publication of his researches there has been no period during which the clinical teachers at Guy's Hospital—Barlow, Rees, and their successors—have failed to insist upon the peculiar characters of the pulse as "hard," "wiry," "resisting," or "incompressible," or (to use a more modern expression) upon the *increase of arterial tension*, which belongs to the affection. But within the last few years the relation between Bright's disease and cardio-vascular changes has been zealously studied by numerous pathologists both here and on the Continent. And although there are still very wide differences of opinion on many questions, a great deal may be stated upon which all, or almost all, observers would agree.

The great point to be insisted upon is that *hypertrophy of the heart* occurs both in "parenchymatous nephritis" and in "renal cirrhosis." It also sometimes develops itself when the kidneys have become atrophied as the result of hydronephrosis, or of some other affection of the renal pelvis, as in cases recorded by Cohnheim. In association with the lardaceous change, however, it is not seen unless the renal cortex has become secondarily affected with inflammatory lesions that have already reached an advanced stage. And it may be comparatively slight, or altogether absent, in persons wasted from phthisis or some other chronic disease, and in those who are very old.

Evidently, therefore, no explanation of the occurrence of cardiac hypertrophy can be valid unless it is applicable to various forms of Bright's disease. The extent to which the heart becomes enlarged differs in different cases—partly according to their duration: and it is far greater in renal cirrhosis than in any other form. Thus, whereas in the early stage of parenchymatous nephritis its weight may range up to fifteen or sixteen ounces, and in the latest granular stage of that affection up to seventeen or possibly twenty-one ounces, there are some instances of renal cirrhosis in which it reaches twenty-three, twenty-four, twenty-five, or even twenty-eight ounces. Traube states ('Ges. Abh.,' iii, p. 239) that he has been able clinically to determine the presence of hypertrophy of the heart within four weeks from the commencement of an acute renal affection. The chamber chiefly affected is the left ventricle, the walls of which (and also the papillary columns of the mitral valve) become extraordinarily thick and fleshy, their substance being made up of muscular fibres of perfectly normal appearance. Sometimes the cavity is of normal size, sometimes more or less dilated. In a good many cases the right ventricle also is enlarged. This probably generally indicates that the left ventricle has not, in spite of its hypertrophy, been able to maintain the circulation efficiently—at least towards the end of life, for I am speaking now of the state of the heart as it is seen post mortem. But to some extent it is inevitable that the right ventricle should share in the process of enlargement, especially when the left ventricle becomes very greatly increased in size.

With regard to the exact character of the changes that take place in the *arteries* there are extraordinary discrepancies in the statements of different observers. In fact, among the pathologists who have especially studied this question within the last few years hardly any two entirely agree in their descriptions. That in middle-aged or old persons affected with Bright's disease the lesions commonly termed atheromatous are often found in the aorta, in the cerebral arteries, and in other arteries of large or medium size, was well known to Bright himself, and also to Wilks, Dickinson, and others who at different times wrote on the subject. But no great importance seemed to attach to this circumstance on account of the frequency with which atheroma is seen in those who are advancing in years. It was Dr George Johnson who in the 'Med.-Chir. Trans.' for 1868, pointed out that the arterioles, not only in the kidneys, but also in the subcutaneous and submucous tissues, in the muscles, and in the pia mater of the brain, become remarkably thickened, and he attributed this change mainly to a hypertrophy of the muscular fibres in their walls. Four years later Sir William Gull and Dr Sutton read before the Royal Medical and Chirurgical Society a paper in which they declared the thickening to be the result of a hyaline fibroid formation, partly outside the muscular layer, partly in the *tunica intima*, the muscular layer itself being often rather atrophied than hypertrophied, and the nuclei of its fibres degenerated. In the course of the discussion which followed, the hyaline appearance described by these observers was shown to be due to the action of the acidulated glycerine in which the preparations were placed for examination. So far as I know Leyden is the only writer who has since laid any stress upon this character; his figures ('Ztschrift. f. klin. Med.,' 1880) represent circumscribed glassy patches lying in the coats of the thickened vessels, and are very unlike those given originally by Gull and Sutton. On the other hand, the different German pathologists who have recently taken up the question likewise fail to confirm Dr Johnson's statements as to the existence of a muscular hypertrophy. Ewald, indeed, admits ('Virch. Arch.,' 1877) that there is an increase in the thickness of the muscular coat, but this, he says, is due to an enlargement of the fibres, and not to a multiplication of them. But according to Sotnitschewsky ('Virch. Arch.,' 1880), when this coat is found thickened, it is as the result of the presence in it of fibrous tissue.

It seems impossible to reconcile these varying descriptions except by supposing that the affection of the arterioles in Bright's disease differs in different cases, perhaps even in the same case at different periods in its course. All recent writers, however, appear to be agreed in considering it as identical with that which was originally named by Friedländer *arteritis obliterans.* But after all, as we shall presently see, it is only with reference to the exclusive theory proposed by Dr Johnson that the exact character of the arterial change is of primary importance.

The view which is to be taken of the relation between Bright's disease and the cardio-vascular changes that I have been discussing, centres upon the explanation of the fact that one of the most marked clinical features of the disease is a state of high pressure or tension in the arteries. This forms an important element in the hard or incompressible pulse to which I have already alluded. Since the invention of the sphygmograph it can be estimated much more accurately than formerly. The first point to be noted in all such tracings is that the pressure applied to the artery while they are being taken is far greater than that which brings out the characters of the pulse most distinctly in health; as registered by Dr Mahomed's spiral eccentric it was from four to six ounces, instead of being from one and a half to three ounces. The next points are the extent of the tidal wave and the distance of the dicrotic notch from the upstroke; these indicate prolongation of the ventricular systole. The last point is the distance of the bottom of the dicrotic notch above the base line (*cf.* p. 62).

If, now, we consider the pulse as it is appreciated by the finger, we find, according to Dr Mahomed, that these characters may be distinguished in it: First, it is *persistent*; even in the intervals between the cardiac beats, the artery feels full; it may even be visibly full and tortuous. One might imagine that its coats were thickened, but on emptying it by pressure above, one finds that it cannot be rolled beneath the finger, as a thickened vessel can be. Next, it is *long*, not falling away as soon as it has reached the finger, but pushing or laboured in character. Lastly, it is *hard* or *incompressible,* requiring much force to overcome it. Both the last characters are really indications of the state of the left ventricle, generally associated with high arterial tension—the slow prolonged systole, and the hypertrophy of the ventricular walls. Conversely, examination of the heart often yields valuable corroborative evidence. There may be an enlarged area of percussion-dulness, displacement of the apex outwards, and a heaving, laboured impulse. It is, however, important to be aware of the fact that these signs are not seldom absent, even when there is no obvious emphysema or other disease of the left lung to account for it. Thus, in cases of renal cirrhosis, when perhaps the patient has been admitted with cerebral hæmorrhage, I have again and again failed to detect any indication of cardiac hypertrophy, although at the autopsy a day or two later the organ has been found enormously enlarged. It is particularly in these cases in which the hypertrophy is unattended with any dilatation that the difficulty arises. On auscultation the first sound may appear to be dull and prolonged, or reduplicated, or even murmurous. Dr Mahomed also maintains ('Guy's Hosp. Rep.,' 1879) that it is sometimes preceded by a short sound having precisely the characters of the presystolic murmur of mitral stenosis. But a more characteristic auscultatory sign, and one which is the direct result of the increased arterial tension, is the loud, ringing, or even metallic quality of the (aortic) second sound, as it is heard at the base of the heart, or over the carotid artery. And sometimes a diastolic shock can be felt by the hand placed over the cardiac region.

With regard to the cause of the high arterial tension in Bright's disease there is much difference of opinion. From the days of Bright down to the comparatively recent time when the question first began to be actively discussed, the view generally accepted was that the altered state of the blood created an obstacle to its passage through the capillaries, and that the heart had consequently to put forth more force to maintain the circulation. In 1868 Dr George Johnson, relying upon his observations as to the existence of a hypertrophy of the muscular walls of the arterioles, propounded the theory that these vessels exert a "stopcock" function, resisting the passage into the capillaries of blood which, as the result of defective elimination by the kidneys, is noxious to the tissues. In fact, he imagined an active antagonism between the heart and the arterioles, as the result of which they each become hypertrophied. I do not know that any subsequent writer has adopted this suggestion, which certainly on the face of it appears highly improbable. More recently Dr Saundby and Dr Mahomed have reverted to the view that the obstruction is in the capillaries. But, believing that the high tension in the arteries precedes the development of renal disease, their notion is that the supposed impurity of the blood is due, not to imperfect excretory activity on the part of the kidneys, but rather to over-eating and over-drinking, by which it becomes charged with injurious matters. The objection to all such theories, however, is that there is no proof whatever that any changes in the circulating fluid are capable of retarding its flow through the capillaries. Physiologists, I believe, admit no causes of such retardation except alterations in the capillary walls themselves; and these only locally, as in the case of inflammation. And although Ustimowitsch and Grützner have shown that in animals the injection of urea into the blood is capable of increasing the arterial pressure, yet this occurs only when the quantity injected is so large as to deprive the experiment of all applicability to human pathology.

Such considerations have led some German pathologists to look elsewhere for an explanation of the cardio-vascular changes in Bright's disease, and of the high arterial tension which is so closely associated with them. Traube, in 1856, suggested that destruction of the renal parenchyma should have two results, each of which might tend to augment the pressure in the arteries; one being the accumulation of water in the blood from impairment of the secretory activity of the kidneys, the other the diminution in the amount of blood flowing from the arterial into the venous system as a consequence of obliteration of vessels in those organs. Now, by the researches of Cohnheim and Lichtheim (see p. 443), it has been shown that the first of these two conditions cannot act in the manner supposed, and the second certainly seems altogether inadequate to produce any marked effect. Cohnheim, however, has recently put this part of the question in an entirely new light. He gives reasons for thinking that the activity of the circulation through the kidneys at any moment—in other words, the state of the smaller renal arteries as regards contraction or dilatation—depends not (as in the case of the tissues generally) upon the need of those organs for blood, but solely upon the amount of material for the urinary secretion that the circulatory fluid happens then to contain. This suggestion has bearings, to which I shall have to draw attention elsewhere, upon the development of hypertrophy in one kidney when the other has been entirely destroyed. But another consequence deducible from it is that when parts of both kidneys have undergone atrophy, the blood-flow to the parts that remain must, *cæteris paribus*, be as great as it would have been to the whole of the organs if they had been intact. But in order that such a quantity of blood should pass through the restricted capillary area now open to it an excessive pressure must obviously be necessary. This can be brought to bear only by the exertion of more than the normal degree of force on the part of the left ventricle, combined with the maintenance of a corresponding resistance in all other districts of the arterial system. And so one can account at once for the high arterial pressure and for the cardio-vascular changes that are secondary to it.

In putting forth this explanation I have used language somewhat different from that which Cohnheim employs, but I think that my view is essentially the same as his. There is not, indeed, any novelty in the idea which forms the basis of it, namely, that the hypertrophy of the heart in Bright's disease is a *compensatory* change, enabling the organism to withstand the consequences of the disease. But what seems not to have been clearly perceived is that a hypertrophied heart cannot effect this result unless it is supported by a resistance in the systemic arterioles proportioned to that which exists in the kidneys. The renal secretion is surely of sufficient importance to justify our supposing that even such great changes as these should occur in order that it may be maintained. One advantage of the quasi-physiological explanation of the high arterial tension of Bright's disease is that it enables us to see that the exact means by which it is kept up may vary in different cases and at different periods of the same case. At an early stage of the parenchymatous affection it can only be by an active contraction of the muscular walls of the arterioles, such as Dr Gowers believes himself to have seen in the retina. And even in the chronic form of Bright's disease this must still play an important part, at least in cases in which a state of low pressure and dicrotism can be induced by the inhalation of nitrite of amyl, as has been shown by Dr Broadbent. But the extensive occurrence of an *endarteritis obliterans* may be supposed in many instances to be also largely concerned in it.

It is obvious that this view which I have been unfolding fits in with the fact that cardio-vascular changes, like those that occur in Bright's disease, may likewise accompany atrophy of the kidneys from hydronephrosis. Nor do I think that there is much difficulty in bringing it into accord with Dr Mahomed's observation, that there are some young persons with presumably normal kidneys in whom the arterial tension is constantly high, notwithstanding that they are in perfect health. Assuming that the urine in such cases is natural in quality and in quantity, one can but suppose that the kidneys are for some reason incapable of secreting such urine, except under excessive pressure. The condition would thus be comparable with the "renal inadequacy" described by Sir Andrew Clark. A very important question is whether it is to be regarded as a warning of the probable supervention at a later stage of Bright's disease, or of endarteritis, and other vascular changes. One of the points insisted on by Sir William Gull and Dr Sutton was, that the arterio-capillary fibrosis which they described sometimes occurred independently of any affection of the kidneys.

7. *Hæmorrhages.*—It is doubtless as a more or less direct result of the high arterial tension of Bright's disease that the rupture of vessels in different situations is to be explained. Thus, I have had, or shall have, to speak of cerebral hæmorrhage, epistaxis, retinal hæmorrhages, purpura, hæmorrhage from the stomach and intestines. As regards epistaxis, Dr Mahomed notes ('Guy's Hosp. Rep.,' 1881) the fact that even when the patient is much blanched by loss of blood the pressure in the arteries may still remain excessive.

8. *Uræmia.*—That the chief symptoms of Bright's disease are in many cases cerebral has been well known from an early period in its history, but there have been wide differences of opinion as to the mode of origin of such symptoms. These differences of opinion, however, have not prevented their being universally termed "uræmic," although the name uræmia, originally (I believe) invented by Piorry, of course implies in strictness the acceptance of the theory that they depend upon an accumulation in the blood of matters that should normally be excreted by the kidneys. The question of the validity of this theory I shall presently have to discuss, but I may so far anticipate as to say that it appears at the present time to afford

the most satisfactory explanation of the facts, so far as they are understood.

The character of uræmic cerebral symptoms varies in different cases. But by far the most frequent form of them consists in the occurrence of seizures precisely like those of epilepsy. Such "*epileptiform*" paroxysms are seen sometimes in patients who are already confined to bed with dropsy or suffering from other effects of Bright's disease; sometimes in persons who have up to that time been engaged in their daily occupations or who may even have seemed to be quite well. Their onset may be either quite sudden or preceded for a few hours or days by headache, drowsiness, vertigo, a strange fixed expression of the face, dragging pains in the extremities, or a transient rigidity of the face, or of the lower jaw, or of a limb. Nausea and even vomiting, again, may be among the prodromal symptoms, or severe dyspnœa, of some hours' duration. E. Wagner also mentions that the pulse sometimes falls to 60 or even to 40 in the minute.

For a description of the paroxysms themselves I may refer to the chapter on epilepsy (vol. i, p. 682), inasmuch as they are identical in every detail with those which characterise that disease, even to the biting of the tongue, the foaming at the mouth, the involuntary discharge of urine and fæces, and the final sleep or stupor, for which is sometimes substituted an attack of violent maniacal excitement. E. Wagner, indeed, says that the initial tonic spasm is sometimes altogether wanting, though he adds that in many cases it is well marked and prolonged. The pupils, he says, are generally dilated and but seldom small, sluggish, or insensible. At Guy's Hospital, from the time of Addison, it has been usual to describe them as being more often contracted or of the natural size, and as usually retaining their sensitiveness to light. The temperature may rise several degrees, reaching 102° or 104°, or even a higher point still. During the coma which follows the convulsions it slowly falls to normal, or even lower; for some days afterwards it may remain as low as 94° or 95°. The pulse is commonly accelerated while the spasms continue; afterwards it returns to its natural rate, or may become slower still, remaining perhaps (as in a case of E. Wagner's) between 44 and 64 for the next fortnight. After the attack has ceased the patient is sometimes dull and depressed for some days. Hemiplegia has very rarely been observed; it might be expected to occur sometimes, as it does after epileptic seizures.

In many cases, before the insensibility has passed off, after one uræmic paroxysm another sets in; and thus twenty or thirty may occur in succession. The disease is then very likely to prove fatal. But even after a series of fits it is not uncommon for recovery to take place, the convulsions ceasing and the patient regaining consciousness, greatly to the surprise of relatives who have given up all hope. A single paroxysm seldom ends fatally; but in 1862 a woman, aged thirty, died in Guy's Hospital within seven minutes from the commencement of uræmic symptoms. She had just eaten her breakfast, when slight spasmodic movements of the arms suddenly set in. She became pale, and her lips and fingers livid; there was foaming at the mouth. The pupils were dilated. The heart at first continued to beat regularly, but its action very quickly ceased.

Sometimes, instead of a uræmic attack having the typical epileptiform character, its symptoms are of a different kind. In the 'Guy's Hospital Reports' for 1839, Addison described one variety as consisting in a "sudden attack of a coma with stertor, or, in other words, *apoplexy*." Probably it is now a result of his teaching, that we at Guy's Hospital have for many years past been very cautious in diagnosing cerebral hæmorrhage in cases in which there was reason to suspect the existence of kidney disease, notwithstanding the well-known frequency with which it occurs under such circumstances.

In discussing the subject of cerebral hæmorrhage, I pointed out that our post-mortem room experience lends scarcely any support to the view that it is liable to be simulated by uræmia, at least when there have been no convulsions (*cf.* vol. i, p. 510). Nor can I call to mind many cases which have recovered, and in which the diagnosis has been doubtful. Dr Roberts cites three apoplectiform cases, but each of them is open to criticism. One patient had had "a few drops" of laudanum given to him for diarrhœa just before the cerebral symptoms set in, so that it may be doubtful whether they were not due to the excessive action which even small doses of that drug are known to exert when the kidneys are diseased; in the second case epileptiform convulsions were present; in the third case there seems to have been no autopsy, so that the possibility of cerebral hæmorrhage is not excluded. Addison was of opinion that the two affections might be distinguished by the characters of the stertor that accompanied them; in uræmia, he said, the sound was more hissing, "as if produced by the air striking against the hard palate, or even the lips, rather than against the velum and the throat, as in ordinary apoplectic stertor." He also said that the respiration was from the first much more hurried. Whether any value can still be attached to these observations I do not know, but I think that few persons would at the present day think of assigning much importance to another criterion mentioned in the same paper, namely, to pallor of the face as contrasted with flushing.

In other instances uræmia is said to manifest itself by *delirium*, lasting for days together, or by rigidity of one or more of the limbs, or, according to Charcot, by tremors like those of paralysis agitans. In a case of Dr Roberts', in which the paroxysms coincided with the catamenial periods, consciousness was not lost; "during the convulsions the patient knew the persons about her, and called loudly to be held fast." Bright, in the 'Guy's Hospital Reports' for 1840, related a case in which for two days before death there occurred a very distressing and almost incessant twitching of the muscles, which increased until the arms and the legs were forcibly drawn up and the face was distorted by the spasms, yet the faculties of the mind were perfect to the last.

But the most remarkable of all the effects of uræmia is, perhaps, *amaurosis*. This not infrequently occurs in association with the epileptiform seizures, the patient, when he recovers consciousness, finding himself blind. Sometimes, according to E. Wagner, it precedes the convulsions. But in the cases that I have seen it has, I think, been the only symptom, unless there was also headache. It sets in suddenly, is bilateral, and is almost always complete, the patient having not the slightest perception of light. Dr Gowers says that the pupils generally still react to light; but E. Wagner says that they are sluggish, and that they may in some cases be altogether insensible and widely dilated. The ophthalmoscope scarcely ever shows any change in the optic discs or retinæ; two instances, however, are cited by Dr Gowers, in which foreign observers are said to have detected slight œdema of the discs, which passed off as the amaurosis was recovered from. For it almost invariably happens that this alarming affection rapidly subsides, the patient regaining sight within twelve or twenty-four hours, or at the longest in the course of a few days. E. Wagner says that such a favourable termination may occur even when the pupils have lost their sensitiveness to light. The fact that, as a rule, the pupils react seems to show that the seat of uræmic amaurosis must be above the *corpora quadrigemina*. In some instances it is said that a transitory defect of hearing, or even complete deafness, has been observed as a sequela of a uræmic seizure.

Lastly, in some cases, uræmia shows itself by much slighter symptoms; by transitory *trismus* perhaps, or by short attacks of *spasm* in some of the facial muscles, or in those of the eyeballs, or of a limb; the patient retaining consciousness, or being at most confused or dull of intelligence.

In striking contrast with these varied forms of what may be termed *acute uræmia* are some which are described as *chronic*. The latter are not, like the former, always obviously cerebral in their character. The lungs or the digestive organs may appear to be the parts affected. But there is good reason to believe that even in such cases the starting-point of the symptoms is generally, if not always, in the brain. When there are cerebral symptoms they usually consist of *headache*, giddiness, or drowsiness, any of which may go on for weeks or even for months continuously, or with intermissions. The patient's aspect is often remarkably dull and expressionless; he lies in bed, taking no notice of what goes on around him, and altogether indifferent to his own condition. Ultimately he may fall into complete stupor. Sometimes the general symptoms are very like those of the typhoid state, the tongue being dry and brown, and sordes collecting upon the teeth and lips. I have mentioned such cases in discussing the diagnosis of enteric fever (vol. i, pp. 202, 203).

In other cases, the principal indication of chronic uræmia is *dyspnœa*. This is said by E. Wagner to be generally paroxysmal, coming on chiefly at night, like asthma. It may also resemble that affection in being mainly expiratory; or, as in a case which occurred at Guy's Hospital in 1865, and in which œdema glottidis was suspected during life, it may be mainly inspiratory, as though there were laryngeal stenosis; or, again, both inspiration and expiration may be free, being simply hurried and deep.

Another sign of uræmia may be an intense *itching* of the skin. Sometimes patients go on scratching or rubbing themselves even when they are so far unconscious that it is quite impossible to rouse them.

One of the most conspicuous symptoms may be *vomiting*, though E. Wagner says it is not so frequent in chronic as in acute uræmia. At first it may occur only in the morning, when the stomach is empty. Afterwards it may take place whenever any food is taken, and may be exceedingly intractable, continuing for weeks or even for months. Urea may often be discovered in the matters rejected, and some have supposed the vomiting to be the result of its presence in the stomach. But in Voit's experiments upon animals it was not found that urea ingested with the food set up vomiting at once, but only when there had been time for it to be absorbed into the blood, and to act upon the nervous centres. The vomited matters are sometimes alkaline, and then some of the urea in them may have been decomposed into carbonate of ammonia. But, as a rule, they are acid. Bartels has suggested that œdema of the walls of the stomach may sometimes be the cause of vomiting in Bright's disease; but, so far as I know, such an affection is rarely observed in the post-mortem room, and to suppose it capable of producing vomiting is, after all, a mere assumption.

Hiccough is not uncommon in association with other effects of uræmia. It rarely occurs by itself; but E. Wagner mentions one instance of chronic Bright's disease in which it and a slight œdema of the lower limbs were the sole symptoms.

Diarrhœa is of rather frequent occurrence, and often accompanies vomiting. It sometimes seems to depend upon an inflammatory affection of the intestinal mucous membrane, which may either be œdematous or ecchymosed, or even in a state of severe diphtheritic inflammation, like that which is met with in dysentery. I have seen its surface covered with large leathery patches, or extensively ulcerated. Such lesions are said by German writers to occur especially in the cæcum and in the ascending colon. In cases of this kind the evacuations often contain blood, and mucus and pus in large quantity. But Bartels says that such matters may be present when the bowel is simply œdematous. There is some doubt as to whether the diarrhœa and the enteritis of Bright's disease should be regarded as uræmic symptoms in a strict sense. Cohnheim and other recent writers think that they are rather effects of local irritation by carbonate of ammonia, produced by the decomposition of urea under the influence of the alkaline contents of the intestine.

Epileptiform convulsions and the other symptoms that have been grouped together under the name of acute uræmia may accompany any form of Bright's disease, but are perhaps especially frequent in the nephritis of scarlet fever, cholera, and pregnancy. I have already stated that they sometimes pass off; and it may be added that their occurrence is no proof that the renal disease that causes them is so severe or so advanced as to be incapable of being recovered from. On the other hand, the prolonged stupor, the typhoid symptoms, and the other phenomena of chronic uræmia probably always end fatally. They are never seen in the more acute forms of Bright's disease—as, for example, after scarlet fever, or in association with pregnancy—unless the so-called "cholera-typhoid" (vol. i, p. 296) is to be taken as an instance to the contrary.

Theory of uræmia.—In discussing the cause of the symptoms that we have thus grouped together under the name of uræmia, we must in the first place consider whether those which are obviously cerebral may possibly be dependent upon actual lesions of the nervous centres. Some years ago Traube propounded the theory that they were due to œdema, combined with anæmia, of the brain. The œdema he supposed to be brought about by the action of a hypertrophied heart upon the smaller intracranial blood-vessels, assisted by a watery condition of the blood itself. But every part of his theory has since been shown to be untenable. In many instances the brain is found after death to be perfectly dry; and when it is œdematous, Bartels is probably right in thinking that this is an effect, rather than the cause, of any convulsive seizures that may have occurred, unless indeed it is a mere accidental result of wasting of the brain, as is doubtless very often the case. Sometimes minute spots of hæmorrhage—"capillary hæmorrhages," they are often called—are found in greater or less numbers in the substance of the brain. I have notes of two instances of Bright's disease, both with large white kidneys, in which the pons and the bulb were full of such hæmorrhagic spots. It seems most likely that they also are produced by the disturbance of the intracranial circulation which cannot but accompany the uræmic paroxysm; their occurrence is far too exceptional to admit of their being regarded as its cause.

We are therefore driven to what may be termed the chemical theories of uræmia. Now, as we shall presently see, there are considerable difficulties in the way of the most natural supposition of all, namely, that it depends upon the retention in the blood of urea or of other constituents of the urine, which the kidneys have failed to excrete. Frerichs consequently suggested in 1851 that the poisonous agent was really carbonate of ammonia formed in the blood by decomposition of urea under the influence of a ferment. Subsequently Treitz amended the hypothesis, supposing that the carbonate of ammonia was produced, not in the blood, but in the stomach and in the intestine, a vicarious excretion of urea into the alimentary canal first taking place, and the carbonate of ammonia being afterwards reabsorbed into the blood. But this theory of an "ammoniæmia," though at one time it was widely adopted in Germany, is now universally abandoned. On the one hand, it has been shown that though carbonate of ammonia, injected into the blood of animals, causes symptoms somewhat like those of uræmia, the resemblance is after all incomplete, and that many other salts produce like effects. On the other hand, it has been denied by many experimenters that carbonate of ammonia can be detected in the blood of uræmic patients, and there appears to be no doubt whatever that, if present at all, it is not in sufficient quantity to account for the effects attributed to it. One clinical point which Frerichs adduced in support of his view was that by holding a glass rod moistened with hydrochloric acid near the mouth of a uræmic patient, the presence of carbonate of ammonia could be recognised in the expired air by the white fumes of chloride of ammonium that were formed. Schottin, however, showed that in many uræmic patients this test completely failed, whereas it often succeeded in other patients who lay in a typhoid state from whatever cause, the carbonate of ammonia being set free from dried secretions within the mouth, instead of being exhaled from the lungs.

Recent observers have accordingly fallen back upon the older and simpler theory, which supposes that the symptoms of uræmia are due merely to the presence in the blood of urea and of other elements of the urine.

The objections which had caused this view to be discarded were drawn partly from experiments upon animals, partly from clinical observations of Bright's disease. Experiments had seemed to show that urea, and even urine itself, could be introduced in large quantity into the blood of animals, without giving rise to any ill-effects. Voit and Oertel, however, have found ('Ztschft. f. Biol.,' 1868) that although urea when added to the food of a dog produces no symptoms so long as it can be freely excreted by the kidneys, yet if the animal is not allowed to drink any water symptoms like those of uræmia appear. The clinical objections to what may be termed the *uræmic* theory of uræmia were taken by Frerichs from a work which had shortly before been published by Dr Owen Rees; they were briefly that the occurrence and the severity of the paroxysms bore no necessary relation to the quantity of urine secreted, and that the blood was sometimes loaded with urea, without any such symptoms appearing.

If now we turn to inquire what are the characters of the urine in cases of Bright's disease at the time when uræmia develops itself, we find that they differ in different cases. As a rule, the renal secretion is very much diminished in quantity for several days before the symptoms set in; it may even be completely suppressed. But sometimes there is a normal flow of urine, which, however, contains much less than the due amount of urea. And sometimes the quantity of urine is rather excessive, as in a case of E. Wagner's, in which the patient, a man named Richter, for three successive days passed seventy ounces daily. In that instance, however, its specific gravity ranged only from 1006 to 1010, so that after all the excretion of urea and of the other solid constituents of the urine probably was defective. The man had had no obvious œdema, and there was therefore no reason for supposing that a reabsorption of dropsical fluid had anything to do with the large amount of urine poured out by his kidneys. E. Wagner, however, remarks that the tissues of the dead body may be found to be distinctly œdematous, when there had been no clinical evidence of it. It seems, therefore, not impossible that the absorption into the blood of such a latent accumulation of fluid may sometimes be the real cause of an excessive flow of urine previous to the development of uræmia, especially as the subsidence of dropsy is known to be frequently followed by uræmic symptoms.

Upon this difficult question of the relation of uræmic symptoms to the excreting action of the kidneys some fresh light has recently been thrown by certain observations made by Fleischer ('Deutsch. Arch.,' xxix, 1881). He instituted careful analyses of the urine passed by persons affected with Bright's disease, comparing them in each instance with analyses of the urine of healthy persons placed under exactly the same conditions as regards diet. He found, as a rule, that the amount of urea excreted by those who had

Bright's disease was much diminished; but when uræmia set in, the amount of urea became increased far beyond the normal, either on the day of the seizure or else a day or two later. The explanation which he suggests is that when the accumulation of urea (and of other urinary constituents) reaches a point at which the system ceases to be indifferent to their presence, so that uræmia results—they at the same time stimulate the heart and the nerves to expel them. Henceforth, therefore, it must be borne in mind that the fact of an abundant elimination of urea taking place during or after a uræmic seizure, is no proof that it may not previously have been deficient, and this deficiency the cause of the seizure.

On the other hand, even when systematic analyses of the urine show that the action of the kidneys has been imperfect, as in the cases in which Bartels found that the amount of urea excreted was reduced to between 130 and 190 grains daily, there is always the further difficulty that in many other cases it is quite as low, without any uræmic symptoms arising. Cases also occur in which the urine is more or less completely suppressed for a week or longer before uræmia develops itself. After all, however, such facts are entirely in accordance with clinical experience in general. The effective operation of all "causes" of disease is liable to be interfered with by unknown conditions, of which the individuality of the patient is perhaps the most important. When describing renal cirrhosis I shall have to point out that uræmia is comparatively seldom met with in persons advanced in years; perhaps this suggests that a "predisposition" on the part of young subjects is one factor in its ætiology.

All doubts as to the occurrence of imperfect elimination by the kidneys in uræmia might be set aside if it were known that the blood in this state invariably contains more urea than in health. The earliest analysis of the blood in chronic Bright's disease seems to have been made by Dr Babington, who states that in a case under the care of Dr Bright himself there was as much urea in the circulating blood as in the urine, a thousand grains of blood yielding fifteen grains of urea! Recent observers, however, have found much smaller quantities than this. E. Wagner says that, instead of the normal proportion of 0·16 or 0·2 parts per 1000, there may be 0·4 or 0·6 parts, or more. He further cites an observation of Hoppe's, who in the blood-serum of a cholera patient with uræmia discovered 1·27 parts of urea per 1000. But elsewhere he says that the quantity of urea in the blood has several times been found to be small, "so that an overloading of the blood with this substance certainly cannot be in all cases the cause of uræmia." It seems to me, however, that before we accept this important conclusion, we ought to know exactly at what period of the disease the analyses have been made in which no excess of urea has been detected. If Fleischer's observations are correct, it seems quite possible that in the course of a uræmic seizure, or afterwards, the blood might contain no excess of urea, and yet that a great excess might previously have been present, and have given rise to the seizure.

Urea may also be discovered in considerable quantity in the various secretions. I have already mentioned its presence in the gastric and intestinal fluids. In one case in which there were bronchitis and extensive pneumonia, Fleischer found it in the sputum to the amount of about thirty grains in the thirty-seven ounces expectorated during twenty-four hours. But the most interesting fact of all is, perhaps, that in some uræmic patients urea is excreted by the skin. This seems only to occur shortly before death, and scarcely ever without the urine being completely suppressed. Schottin first observed it in 1852 in cholera patients. The 'Guy's Hospital Reports' for 1874 contain a report, by Dr Frederick Taylor, of a patient with Bright's disease, in whom, two days before death, there appeared on the face, neck, and hands white masses which adhered pretty firmly, and which, when removed, were found to be irregularly shaped, with crystalline spiculæ and prisms projecting from them. They yielded the several reactions of urea. The patient's face is described as having looked as though flour had been sprinkled over it. In some other cases the appearance is said to have been just as though a lather of soap had been allowed to dry on the surface, or as though the beard were frosted.

All recent writers on the subject endorse an opinion which was first expressed by C. Voit ('Zeitschft. f. Biol.,' 1868), namely, that uræmia is not due to the poisonous action of any one ingredient of the urine—whether urea, uric acid, kreatinin, or other extractives. Voit himself was inclined to attach a considerable share of the uræmia to the salts of potass. In birds and in reptiles, which form no urea, he was quite ready to admit that the retention of the uric acid which they should excrete may be an adequate cause of precisely the same symptoms. In short, he believed that they may be produced by "any substance which is not a normal constituent of the body if it accumulates in large quantity and is not eliminated."

Such a conclusion, however, is opposed to one clinical fact, for a clear recognition of which we are indebted to Dr Roberts, and which seems to me of the highest importance both to the physiologist and to the physician. It is that symptoms altogether unlike those of uræmia, and holding a completely different course towards a fatal issue, are presented by cases in which the failure to eliminate urea and the other ingredients of the urine is absolute, but in which the cause of the suppression of the renal secretion is not an affection of the cortex of the kidneys, but obstruction of the ureters. As for the clinical characters of "obstructive suppression," I have discussed them fully above (p. 387). Here I have only to insist on the significance of the circumstance that such a distinction exists. It seems clearly to show that where there is healthy kidney-substance, with an active blood circulation through it, the waste products that should be excreted in the urine undergo some chemical changes that render them incapable of producing uræmia, notwithstanding that they are retained in the body. Nor are the results of experiments upon the lower animals, so far as I can learn, inconsistent with this view, though they lend it but little support. Obstructive suppression is, of course, easily produced by ligature of the ureters; the effects of this operation are spoken of as identical with uræmia, but it is perhaps not to be expected that distinctions between different groups of symptoms should be so obvious in animals as in man. On the other hand, it is by no means easy to bring about non-obstructive suppression in such a way as to afford a satisfactory comparison. There are two ways in which one ought to be able to annul the activity of the secreting substance of the kidneys. One is by ligaturing the renal arteries, the other by excising the kidneys. Now, ligature of the renal arteries has been shown by Herrmann ('Sitzungsber d. Wien. Acad.,' 1861) not to be effectual in arresting the blood-supply to the kidneys, which may continue to pour forth urine. And excision of the kidneys is an exceedingly severe operation, very apt to produce vomiting and other ill-effects that make it unfair to contrast it with so simple a procedure as ligature of the ureters. It is, however, an acknowledged fact that after excision of the kidneys the quantity of urea which is found in the blood is not nearly so great as after ligature of the ureters. Salkowski points out that the cause of this may very well be the vomiting that follows the former operation. But it surely is at least possible that it may in part be due to the fact that in normal circumstances a part of the urea excreted in the urine is formed by the kidneys, as has been supposed by Oppler and by others. Dr Roberts, indeed, boldly maintains that the cause of uræmia is the accumulation in the blood of products intermediate between urea and the albuminous substances from which it has its origin. As examples of such intermediate products he alludes to kreatin, kreatinin, and other extractives; but, according to other views with regard to the formation of urea they would have to be sought for in the amido-acids, namely, leucin (amido-caproic), aspartic acid (amido-succinic), and tyrosin.

In conclusion, there seems to be no doubt that uræmia is produced by the poisonous action upon the nervous centres of materials accumulated in the blood as the result of defective excretion by the kidneys. But it is still uncertain whether this action is excited by one substance in particular, or by all of them together. In many cases the actual outbreak of convulsions is immediately due to some obvious disturbance of the balance of the bodily functions, which may then be supposed to have been previously unstable. Thus Bartels relates a striking instance in which the production of profuse sweating in a dropsical patient by a hot bath, followed by hot packing, at once brought about a series of uræmic attacks; next day the dropsy was gone. Sometimes, perhaps, what directly causes uræmic symptoms is the sudden failure of the heart to keep up an active circulation through the renal vessels, so that the excretory function of the kidneys, which may for a long time have been more or less impaired, now becomes rapidly altogether ineffectual.

We must now pass on to consider the several forms of Bright's disease enumerated at pp. 431, 432.

I. Parenchymatous Nephritis.—(*Tubular nephritis—Desquamative nephritis.*) As I have remarked at p. 430, I adopt this name of Virchow's as the most suitable that I can find for the purpose, but not as meaning strictly that the pathological changes in the form of Bright's disease that I shall describe under it are limited to the secreting cells of the renal cortex. On the contrary, I shall have to point out presently that the glomeruli and even the connective tissue round them are in many cases markedly affected.

Ætiology.—Parenchymatous nephritis is often due to obvious causes. It may be definitely traceable to *cold.* Bartels cites three well-marked examples of this; one is that of a patient who was taken ill as the direct result of going to sleep half undressed by an open window on a winter's night, after having spent the evening in dancing; another is that of a man who, while perspiring freely, left his smithy and went out into the open air in his shirt, getting wet through with an icy rain; the third is that of a person who was skating, when he broke through the ice, and had much difficulty in extricating himself. Very many cases arise from *scarlatina.* In other cases, again, no definite cause can be found. It is not improbable that some of them are really due to a latent attack of scarlet fever, particularly during childhood. Cholera, relapsing fever, erysipelas, enteric fever, smallpox, measles, all more or less frequently give rise to albuminuria and to nephritis, but it seems doubtful whether the renal affection in any one of these diseases ever assumes a character sufficiently independent to justify our speaking of it as Bright's disease. In women, *pregnancy* is often the cause, especially in primiparæ, and, above all, when there are twins. Sometimes the disease appears to recur in successive pregnancies. It generally manifests itself during the later months of gestation. How it is brought about is not at all clear. It certainly is not due to pressure by the gravid womb upon the renal veins; and the most plausible view seems to be that it results from the kidneys having extra work thrown upon them in the elimination of effete matters. Its onset is often insidious, but those writers who separate acute from chronic Bright's disease include it under the former head. Among the causes that are more or less slow in their action, I should be inclined to place indulgence in *alcohol* as a very important one, although such an opinion is disputed by some observers. Several instances occur to my memory. One is that of a solicitor, usually moderate in his habits, and now (I believe) in good health, who some years ago acquired a temporary albuminuria as the result of drinking sherry in large quantities to induce sleep at a time when he had a great trouble weighing upon him. And I have had another patient whose urine was for many months albuminous, apparently as the effect of habitual excess in stimulants; he changed his ways, and two years later no evidence of any kidney affection could be discovered, and he seemed to have regained his usual health. In countries in which *ague* is endemic, it is said to be a frequent cause of chronic Bright's disease.

Anatomy.—The appearances presented by kidneys affected with parenchymatous nephritis differ a good deal in different cases, even at the same period of the disease, Should it prove fatal during the first two or three months, they are either found of nearly natural size, or more often enlarged up to twice their natural weight. It may be noticed that they are rounded in shape, stretching their capsule, which is not thickened, and can be as easily stripped off as from a normal kidney. Their colour is usually a dull greyish-red, or a paler grey, the pyramids having a much deeper reddish-purple tint. Sometimes, especially if there has been complete suppression of urine, and if death has been due to convulsions attended with great pulmonary congestion, all parts of the kidneys are found gorged with blood, and of a dark chocolate colour. In most cases red points are scattered over the cortex; some of them are blood-filled glomeruli, others are punctiform hæmorrhages. But in some instances, particularly among those attributable to scarlet fever, the kidneys show scarcely any deviation from their natural appearance.

Until within the last few years it has generally been stated that the principal morbid change affects the convoluted tubes of the cortex, the epithelium of which was described as first becoming cloudy and granular, and afterwards proliferating, so as to fill them with masses of irregular or rounded cells. Recently, however, special attention has been paid to the histology of scarlatinal cases by Klebs, and in this country by Klein and by Greenfield; and these observers are agreed that the most constant lesions are those that concern the glomeruli and their capsules. Not only do the nuclei of the capillary tufts of the glomerulus proliferate, but there is also an abundant growth of nuclei within the capsule, leading to adhesion between it and the glomerulus, and ultimately to compression and atrophy of the latter. The peri-glomerular connective tissue also becomes crowded with nuclei, which ultimately develop into fibroid tissue, and the afferent vessel of the glomerulus, as well as its capillaries, undergoes a peculiar hyaline change. Sometimes these lesions are limited to a few of the glomeruli only; sometimes they are very widespread. It is obvious that the obliteration of the space naturally existing between the tufts and the capsule that encloses them must completely abolish the functions not only of the glomerulus itself, but also of the whole length of the convoluted tube that corresponds with it. And, further, the changes in the tufts themselves may fairly be supposed to obstruct the blood-supply to the convoluted tubes, and to affect the nutrition of their epithelium. Consequently, some pathologists are now disposed to see in the "glomerulo-nephritis," as they term it, the fundamental morbid process that follows scarlet fever, and to regard the lesions of the tubal epithelium as secondary and relatively unimportant. If these views should be confirmed in their entirety, the name of parenchymatous nephritis may ultimately have to be given up. At present, however, it is uncertain whether the observations made with regard to the early stages of the Bright's disease consecutive to scarlet fever apply also to that which arises from cold or which is attributable to pregnancy. The occurrence of glomerulo-nephritis is certainly not peculiar to scarlatinal cases, for Cohnheim says that he met with a typical example of it in the case of a man who died some weeks after having his skin rubbed all over with petroleum for four days consecutively.

In cases that have been of somewhat longer duration, the kidneys present appearances deviating still more markedly from the normal. The cortex has now an opaque white or whitish-yellow colour, both on its surface and on section; and this contrasts very strikingly with the red colour of the pyramids. The organs are sometimes very large, much more so than are ever the reddish-grey or chocolate-coloured kidneys of an earlier period. I have notes of three cases in each of which the weight of a pair of kidneys was twenty-eight and a half or twenty-nine ounces, and in one instance Dr Moxon found them weighing within half an ounce of three pounds. On the other hand, it sometimes happens that precisely similar kidneys, still retaining their smooth surface, are of natural size, or even slightly smaller; but such cases are rare exceptions. The shortest time within which I have seen the kidneys assume the "large white" character is five or six weeks; more often the disease has lasted several months. In each of two cases in which there was very great enlargement the patient had had it for fourteen months. On the other hand, I have met with one instance in which the kidneys were still of a brick-red colour at the end of six months. It is to be remembered that a considerable proportion of cases of parenchymatous nephritis fatal at this stage are of insidious origin and slow development, so that their duration cannot be fixed. And, as Cohnhein observes, a very large number of cases that have commonly been classed as examples of the "large white" variety of Bright's disease ought really to be set aside as belonging to the lardaceous form. It cannot be too strongly insisted on that kidneys from the bodies of syphilitic patients, or of those who had phthisis or other suppurative affections, should never be set down to parenchymatous nephritis, until the absence of the lardaceous change has been determined by microscopical examination. I have, however, myself met with one or two instances in which, although there was tubercular disease of the lungs, no reaction with iodine could be obtained.

Histologically, the most striking appearance in most specimens of the large white kidney is the accumulation of immense quantities of fatty epithelial cells in the renal tubes. It is this that gives the opaque yellow colour with reflected light; in thin sections, viewed by transmitted light, the tubes often appear quite black. The stroma of the cortex also is closely studded with fat-granules. Hence such kidneys are sometimes spoken of as "fatty," but very improperly, since that designation ought to be reserved for cases in which a fatty change is the principal one, as in obese persons who also have fatty liver, or in cases of poisoning by phosphorus. Again, it must not be assumed that all large white kidneys are fatty. In those which are translucent and greyish white the amount of fat is often very small.

Dr Greenfield, in his *résumé* of renal pathology in the Sydenham Society's 'Atlas,' speaks of cases, especially in pregnant women, in which the microscope shows that the lesions are almost entirely "interstitial." One distinguishing character of such kidneys is their toughness, almost like that of caoutchouc, contrasting strongly with the soft pulpy texture of those in which the principal changes affect the tubal epithelium. But the fact is that in almost all, if not in all, cases of advanced "parenchymatous" nephritis, interstitial lesions are really present to a greater or less extent. This has been especially insisted on by Weigert, who says that he has for years vainly sought for a specimen altogether free from interstitial changes. They are not uniformly diffused through the renal substance, but (as in many other forms of Bright's disease) consist of patches of nuclear growth, afterwards developing into tracts of connective tissue, in which the nuclei are less numerous. The glomeruli also have their capsules thickened, and pass through every stage in the process of conversion into structureless globular cysts.

Finally, if the disease runs on long enough, the kidneys become shrunken, small, and granular on the surface, though they still (and perhaps always) retain more or less of the whitish-yellow colour. This continues to distinguish them from kidneys affected with the cirrhotic form of Bright's disease; but, as I shall more than once have occasion to remark, the distinction is lost in those cases in which organs primarily cirrhotic become the seat of secondary parenchymatous changes. The occurrence of a granular stage as the ultimate issue of nephritis arising from scarlet fever or of any other cause that commonly produces a large kidney, was at one time denied. But it has now been clearly established, and a typical example of it is figured in Plate 3 of the Sydenham Society's 'Atlas of Pathology.' The case was that of a girl, aged ten, who, rather more than two years before her death, became dropsical as the result of scarlet fever. After four months she recovered, but a year later the face again began to swell from time to time, and she died at last with cerebral symptoms. At the autopsy the kidneys were found to be very small indeed, with thick opaque capsules, hard, tough, and puckered on the surface, presenting on section yellowish-grey masses. In the records of post-mortem examinations at Guy's Hospital I find a good many cases, in young subjects, of a more or less similar kind, the weight of the pair of kidneys being from four to eight ounces. In most of them the history affords no clue as to the date at which the disease had begun. But in some instances it is recorded that there had been an illness, attended with dropsy, several years before death. A case in point, occurring in a young woman of twenty-four, a patient at the London Hospital, is figured in the same Plate of the 'Atlas.' She was said to have been ill for only three months; but her kidneys were found by Dr Sutton to be reduced to about half their normal size, and to be "very granular, and of a reddish colour, everywhere mottled with a yellowish or purplish or greyish substance."

Symptoms.—This form of Bright's disease varies in its mode of onset in different cases. When it is due to scarlet fever, or to a definite exposure to cold, it may begin with a rigor and with well-marked pyrexia. More commonly, the earliest indication of the patient's illness is the occurrence of dropsy. This may first appear in the loose tissue round the eyes, and in slight cases it is especially noticeable before the patient gets up in the morning. Generally, however, it also affects the limbs; and a favourite seat for it is the lower part of the back, as far down as the sacrum. The external genitals, too, are very apt to become swollen, and the prepuce is apt to be so stretched and twisted as greatly to interfere with the act of micturition. In extreme cases almost the whole body is distended with fluid, so that it has a bloated appearance, while the extreme anæmia which rapidly develops itself gives the skin a pale yellow, wax-like colour. The appetite may be bad, the tongue furred, and the bowels constipated. Vomiting is often a marked symptom and there may be much headache. Pain in the loins is often altogether absent, but sometimes it is severe, radiating to the groins and to the thighs, and being accompanied with tenderness to pressure.

The urine, in the early stage, is always scanty, and sometimes entirely suppressed. The patient may be constantly striving to micturate, and yet a few drops of blood-stained liquid may be all that there is to pass. Complete suppression, if it continues for any length of time, is a very grave symptom, and generally points to a fatal issue, which is seldom long delayed, even if the kidneys should afterwards, to some slight extent, resume their functions. When some ounces of urine are secreted daily, it is commonly of high specific gravity (1025—1030) and of dark colour, more or less red or brown from the presence of blood. Its appearance may often be fairly compared with that of strong tea or of porter. It is turbid, and throws down a chocolate-coloured deposit, containing altered blood-discs, swollen epithelial cells, and casts, some of which are hyaline, while others may themselves be full of blood-discs or beset with epithelial cells.

Albumen is of course always present in the urine when there is blood. But the quantity of albumen is often not so large at this period as it is a little later when the hæmaturia has passed off. My colleague, Dr Mahomed, has described, in the 'Med.-Chir. Trans.' for 1874 what he terms a *pre-albuminuric* stage, in which while no albumen can be detected, the guaiacum test reveals the presence of colouring matter of blood and the sphygmograph shows a marked increase of arterial tension. Bartels also mentions cases in which, when scarlatinal dropsy first set in, the urine, though exceedingly scanty, was non-albuminous, and he cites a case of Henoch's in which no albumen could at any time be detected except on the day before death, when the patient was cyanosed and almost pulseless as the result of an attack of convulsions. Ordinarily the amount of albumen ranges from 2 to 5 per cent. The total quantity passed in the twenty-four hours is said to be from 80 to about 400 grains. The excretion of urea is greatly diminished, falling to half the normal amount, or even much less.

When parenchymatous nephritis ends in recovery in the course of a few weeks—as occurs in the large majority of scarlatinal cases, as well as in many of those due to other causes—the dropsy and the other symptoms subside, and the urine gradually recovers its normal characters. It is more abundant, of lower specific gravity, paler; it no longer contains blood, which, indeed, is generally present only at the very commencement of the attack; the quantity of albumen in it becomes less and less, until at length there is none.

On the other hand, when the disease runs on for months, the dropsy continues, or becomes worse. So bloated and anæmic is the appearance of the patient that he is sometimes spoken of as having a "large white" body, as a fitting receptacle for kidneys of the same character. He lies helplessly in bed, his back propped up with pillows, his legs stretched stiffly out before him, or supported by a pillow beneath the knees. The swelling of the external genitals is often extreme. The scrotum looks like a bladder full of water, and is so large that there is no room for it between the thighs; the prepuce is described by Bartels as "curled up like a posthorn." Sometimes the cuticle over some of the distended parts cracks, and the dropsical fluid oozes out in such quantities as to soak through the bedding. This may cause considerable tracts to become excoriated, and ultimately to be covered with pale granulations which, when they skin over, give the surface a warty appearance. Or inflammation may set in, attended at first with a crimson redness, like that of erysipelas, and leading to more or less extensive gangrene of the skin and even of the subcutaneous tissues.

In this more advanced stage of parenchymatous nephritis the state of the urine is very variable. It may still be scanty and of rather high specific gravity. But more frequently it gradually becomes abundant and pale, and then its specific gravity falls considerably below the normal, to 1010 or 1005, or even lower. It may still be highly albuminous; indeed, the quantity of albumen in it may reach 5 per cent., or even more, so that on the application of heat the whole contents of the test-tube set into a solid mass.

On the other hand, as disease progresses this amount of albumen often lessens considerably. Blood in small quantity may be present from time to time, but this is rather exceptional. Casts are commonly found in abundance, some of them hyaline, others containing leucocytes, epithelium, and fat-granules, others completely opaque and granular. The excretion of urea is at all periods of the disease much below the normal amount. Even when the flow of urine becomes more abundant, the total quantity of urea contained in it in the twenty-four hours, instead of increasing, is commonly still less than before. Albuminuric retinitis, in all its forms, is of frequent occurrence; as to its characters I have nothing to add to what has been already stated.

Event.—In some cases of parenchymatous nephritis, at whatever stage of the disease, death occurs as the result of the intensity of the dropsical and other symptoms. Or it may be due specially to the accumulation of fluid in the great serous cavities of the chest, or (though very rarely) to œdema of the larynx. In many instances it is the result of œdema of the lungs, or of pneumonia which has commonly more or less of an œdematous character. In others it is brought about by the supervention of acute peritonitis or pleurisy or pericarditis. Many cases, again, end by uræmia. And in some, perhaps, failure of the heart, as the result of dilatation of the left ventricle, is the direct cause of the fatal issue.

It can but rarely happen that complete recovery takes place when the disease has lasted many months, still less when it has been prolonged for years. But it is surprising how symptoms will sometimes subside and disappear, and how, even after the patient has been waterlogged and has had alarming uræmic attacks, he may regain what appears to be a fair state of health. As an instance of the removal of an albuminuria of long standing by treatment I may cite the case of a medical man, aged twenty-six, whose urine after an attack of scarlet fever was continuously albuminous after meals for more than six years. By the advice of Dr Johnson he was strictly dieted, and at the end of nine months the urine became normal, and remained so eighteen months later ('Brit. Med. Journ.,' 1879, ii). In that case, however, the general health remained good all along.

II. Lardaceous, Waxy, or Amyloid Disease of the Kidney.—From the time of Rokitansky the lardaceous affection of the kidneys has been described as one of the forms of Bright's disease, and Wilks has pointed out that there is good reason for supposing that one of the specimens figured by Bright himself as a "large white kidney" was lardaceous, inasmuch as the liver of the same patient, which is still kept in the museum of Guy's Hospital, presents that morbid change. For a long time the view which prevailed with regard to the relations between lardaceous kidneys and those affected with diffused inflammatory lesions was rather that they were clinically allied as all alike giving rise to albuminuria and to dropsy than that there was any close pathological connection between them. This view doubtless received support from the fact that in the liver and in the spleen the lardaceous change is certainly unconnected with diffused inflammatory lesions; but, perhaps, it has hardly been enough kept in mind that even in other circumstances neither of those organs is liable to such lesions. Of late years, however, all pathologists have perceived that the true reason why it is impossible to exclude the lardaceous kidney from Bright's disease is, that the lardaceous change is so commonly associated in the same kidney with the lesions belonging to the other forms of this disease. With regard to the nature of the association in question I shall have much to say presently. Whatever its nature, this association gives to the lardaceous affection of the kidneys a clinical importance which does not belong to the like affection of any other organ. And therefore the general bearings of that morbid change may be better discussed here than in any other part of the work.

The first real step in the study of lardaceous disease was the discovery by Meckel of the fact that the tissues affected give a peculiar reaction with iodine, turning of a walnut or mahogany-brown colour, whereas healthy tissues remain pale yellow. Virchow further stated that on the addition of dilute sulphuric acid a more or less distinct blue or purple tint can be detected, to which he attached great significance, regarding it as an indication that the lardaceous material was chemically related to principles of vegetable origin, such as starch and cellulose. But many observers have failed altogether to obtain any blue tint, and I think that most will agree with Ziegler that this part of the reaction is generally imperfect. In reality, however, what now alone lends to it any interest is the historical fact that it caused Virchow to give to lardaceous disease in general the name of "amyloid." In Germany this name is still applied to them, though in England it has never altogether replaced the older term, which seems to me to be far preferable as embodying no erroneous theory. For it has long since been shown by Kekulé, and afterwards by Kühne and Rudnett, that the "lardacein" (as it is now called) material contains nitrogen, and is really allied to albumin.*

It is best to employ an aqueous solution of iodine and iodide of potassium as a test for the lardaceous material. The cut surface of the organ to be tested must be first washed free of blood, which, if present in large quantity, makes it impossible to speak with any confidence as to the presence or absence of the reaction. The surface is then lightly brushed over with the solution. A very considerable degree of lardaceous change becomes apparent in a few seconds, by the formation of brown or black spots or streaks. If, however, the change is very slight in amount, it may be perceptible only with the microscope, and after the application of the iodine to a thin section. In 1875 Jürgens made known in 'Virchow's Archiv' the fact that one of the aniline colours—a violet prepared by the action of iodide of methyl upon aniline, though the presence of iodine in the compound is said to be doubtful—gives a beautiful colour-reaction with lardaceous tissues. Those parts which present the lardaceous change slowly become red, whereas the unaltered parts are stained blue. The main advantage of this over the iodine test appears to be the greater definiteness with which the reaction remains limited to certain elements in a complex structure. Thus it is invaluable in determining whether the secreting cells of an organ, or the adjacent capillaries, are the seat of the morbid change. Another important point is that it enables permanent preparations to be made and preserved in glycerine. On the other hand, it cannot be used as a rough test in the post-mortem room; and even as applied to microscopical sections of the tissues it seems to be in no way superior to the iodine test when the question is merely as to the presence or the absence of the lardaceous change in an organ, or even as to its amount.

Without the addition of any colouring matter, the lardaceous change is plainly recognisable with the microscope in thin sections of an organ. The affected parts of the tissue have a swollen, homogeneous, glistening appearance, which cannot be mistaken by an experienced eye.

As to the nature of the process by which lardacein is substituted for the natural proteids of the tissues nothing is as yet known. Whether this material arises *in loco* as a "degeneration" of the normal structures, or whether it is a "deposit" or an "infiltration" derived from the blood, seems to me to be quite uncertain, though most writers on pathology express a confident opinion, one way or the other. In the blood itself it cannot be detected. A point which is in favour of its local origin is the fact that hyaline casts that have been retained within the tubes of the kidney sometimes give the reaction with iodine, though, as a rule, no such reaction can be obtained from them. One can hardly doubt that a chemical change occurs in the albuminoid substance of the casts subsequent to their formation. What makes the question a peculiarly difficult one is that in every organ the parts most apt to become lardaceous are not the proper elements of its structure, but the capillaries and the small arteries. On the "infiltration" theory, it is extremely hard to understand why the change should be limited to the vessels in certain organs, and not equally distributed over those of the entire body. Moreover, even in a single organ—such as the kidney—it is apt to be very partial, occurring only in certain glomeruli, or only in some loops of the same glomerulus. Surely this looks more like a local "degeneration." But, on the other hand, it may be urged that there are obvious analogies between the lardaceous change and the process of calcification, which of course is due to the deposition of lime-salts from the blood.

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* [The earliest term applied to this condition was "lardaceous" (*speckartig*, like bacon-rind) by Rokitansky and the Vienna school. In Edinburgh it was named "waxy" by the late Prof. Sanders. Budd and other English writers called it "albuminous." Any of these is better than the misleading term "amyloid" (starch-like); but lardaceous is used in France and America, and is adopted in the nomenclature of the College of Physicians.—Ed.]

Within the last few years the lardaceous change has from time to time been met with in strange situations, where its occurrence certainly would not have been anticipated; and what is still more remarkable is that then it has often been absent from its usual seats. I may cite, for example, Burow's case of lardaceous degeneration of laryngeal tumours, Ziegler's case of lardaceous nodules at the base of the tongue, Birch-Hirschfeld's statements as to the presence of lardaceous material in mesenteric glands after enteric fever, and, above all, the curious examples of a lardaceous change in the vessels of the conjunctiva recorded by Sämisch, by Leber and by Kyber. References to all these observations may be found in a paper by Kyber in vol. 81 of 'Virchow's Archiv.' It has also been shown that by carefully searching the various structures of the body, even when the lardaceous change has its usual distribution, one can often detect its presence in a good many other parts besides those in which it is commonly recognised. But for practical purposes, and as an important pathological process, it may be fairly regarded as limited to a small number of organs, these being the kidneys, the liver, the spleen, the intestine, the adrenal bodies, and the thyroid. And in the last two of them, so far as is at present known, it gives rise to no symptoms and has no clinical significance whatever.

Anatomy.—The appearances presented by a LARDACEOUS KIDNEY vary widely in different cases. Sometimes the lardaceous affection of its vessels is the only change in the organ discoverable, whether by the naked eye or with the microscope. So far as I have seen, however, this is only the case when the immediate cause of death has been some other disease; generally one of the various maladies to which, as we shall presently find, the lardaceous change is commonly secondary. I do not remember to have met with an instance in which, at this stage of the renal affection, the patient has succumbed to cachexia from the simultaneous development of the lardaceous change in several organs. And how rare it is in the post-mortem room for a kidney to be seen which presents no other lesions than the lardaceous change, may appear from a collection of more than sixty cases of lardaceous kidney which I have taken from our records at Guy's Hospital; among them there seem to be only three or four uncomplicated examples.

A kidney thus affected is of the natural size and looks healthy, except that a keen eye may perhaps perceive the glomeruli to be somewhat more distinct and more translucent in appearance than is natural. On the addition of iodine the change is generally most marked in the glomeruli; and sometimes it is entirely limited to them. Nor does it affect all of them at once, being on the contrary often confined to a few of them only, and even in the same glomerulus to one or two of the loops of which it is made up. Not unfrequently the afferent arteries are involved as well, their middle coat being the first part to suffer. But sometimes the reaction is obtained, not in the cortex, but in the straight vessels of the pyramids. And sometimes it seems to begin in both the cortex and the pyramids simultaneously. When the change is very far advanced, the capillaries round the renal tubes are also affected, and even the basement-membrane of the tubes, and perhaps the epithelium lining them. But I should doubt whether changes so extensive are ever seen in uncomplicated or "simple" cases.

In the immense majority of instances, lardaceous kidneys are the seat of other lesions likewise, by which they are greatly increased in size. In about a quarter of my cases I find that the weight of the two organs together has been from twenty to twenty-seven ounces. The appearance of such kidneys is sometimes very peculiar, and fully justifies their being termed *waxy*, or (in Germany) *butter-kidneys.* Their surface is smooth, pale yellow, with conspicuous stellate veins; their section is shining and polished, of a semitranslucent grey or yellowish colour, sometimes showing obviously the grey swollen glomeruli, sometimes mottled more or less thickly with creamy opaque spots and streaks, where fatty granules are present in abundance. What causes the enlargement of the organs in these cases is undoubtedly the accumulation of inflammatory products, not only in the tubes, but also in the interstitial tissue. But as to why such a diffused nephritis should arise there have been all possible differences of opinion.

Cohnheim is inclined to believe that it is an independent result of the same cause which produces the lardaceous change, and which we shall presently find to be generally either syphilis or prolonged suppuration. But I think that the association of the two morbid processes occurs far too frequently to admit of such an explanation, especially as I suppose that no pathologist will declare a diffuse nephritis, apart from the lardaceous change, to be a common result of such causes. The known ætiology of the lardaceous change clearly excludes the view of Cornil and Ranvier that it is preceded by the nephritis. We have therefore no alternative but to suppose that in some way lardaceous degeneration must cause the nephritis. And the objection that no similar inflammatory process is found to arise in a lardaceous liver or spleen loses all its force when one remembers that neither of these organs is subject, under any circumstances, to an affection comparable with Bright's disease.

Weigert observes that the peculiar position of the glomeruli, between the arterioles of the renal cortex and the capillary blood-supply to the tubes, may well render any obstruction to the flow of blood through the glomeruli the cause of damage to the tissue beyond. He believes that the first result of the lardaceous change is to produce fatty degeneration of the epithelium, and that this degeneration, whatever its origin, is apt to set up diffused interstitial nephritis.

Sometimes lardaceous kidneys are found after death to be smaller than natural. This occurred in about one out of five or six of the cases that I have collected from the post-mortem records of Guy's Hospital. As a rule the loss of size was not very obvious, the two organs weighing together not less than seven and a half or eight and a half ounces; but in one instance the weight (of what happened to be a "horseshoe" kidney) was only three and a half ounces. There was always, however, much irregularity and shrinking of the surface—a more or less markedly "granular" condition; and the loss of substance was no doubt far more considerable than was indicated by the weighing machine, inasmuch as they still retained large quantities of inflammatory exudation, as well as the lardaceous material itself. Such wasted organs clearly represent the most advanced stage of the lardaceous affection, and it is fair to assume that its course has been more than usually slow and protracted. But there is no evidence that kidneys that ultimately become thus reduced in size have passed through an earlier stage in which they were enlarged to any great extent. Some observers have tried to account for such cases by supposing that the kidneys were already contracted and granular (from gout or some other cause) before they began to be affected with the lardaceous change, as the result of syphilis or protracted suppuration. And there seems to be no doubt that in 1866 we had at Guy's Hospital an instance of the accidental association of the two morbid processes in the case of a woman who died at the age of forty-five of cerebral hæmorrhage, and whose kidneys, besides being lardaceous, were granular and full of minute cysts. Weigert remarks that there ought not, with the microscope, to be any difficulty in distinguishing mixed cases of this kind, since the glomeruli would doubtless be shrunken and atrophied after the manner described at p. 476 *infra*, whereas in the most advanced stages of a lardaceous kidney they continue to present the appearances characteristic of that affection. The hypothesis of an independent granular change could, however, be hardly applied to two of my cases, each of which occurred in a young man of twenty-four, for at that age such a change in the kidneys is almost unknown; in one of these cases the organs weighed seven and a half, in the other six and a half ounces.

Symptoms.—The *characters of the urine* secreted by lardaceous kidneys were first studied by Traube in 1858; and to his account of them not much has been added by the labours of Grainger Stewart and others, who have since taken up the subject. But although cases doubtless sometimes occur, in which the state of the urine alone may suggest to an experienced observer the idea that the renal affection from which the patient is suffering is lardaceous, and not one of the other forms of Bright's disease, such cases are, I think, exceedingly infrequent. Indeed, considering how constantly the lardaceous change in the kidneys becomes associated with other lesions, and how diverse these lesions may be, it would be absurd to suppose that the urine should always present the same characters. The cases in which the influence of the lardaceous affection should be most clearly traceable are, of course, those in which it is uncomplicated with inflammatory changes in the renal cortex. But there is much force in Cohnheim's remark, that the existence of such "simple" lardaceous kidneys can scarcely ever be determined, except in patients who have fatal disease of other organs which causes them to be wasted and anæmic, and to suffer from pyrexia, diarrhœa, and other symptoms that must in themselves disturb the balance of the nutritive changes in the body. Thus Bartels appears to be right in declaring that there is no evidence that the lardaceous change in itself interferes with the excretion of urea; if this is diminished, it is a result of a general lowering of the activity of the chemical processes throughout the body. Indeed, the effect of this change in the glomeruli appears rather to be that their walls are rendered more permeable than before.

With regard to the *quantity* of urine secreted by lardaceous kidneys, different writers make very different statements. According to Grainger Stewart, unless an extreme degree of nephritis is present, it is excessive, ranging from fifty to two hundred ounces daily. He also maintains that an increased flow of urine is in many instances of great clinical importance as an early symptom of the lardaceous affection, preceding albuminuria. Bartels, however, gives much smaller average amounts—from fifty to sixty or seventy-six ounces—and he expressly remarks that there is never such a degree of polyuria as occurs (for example) in some cases of granular disease of the kidneys. On the other hand, E. Wagner declares that in the majority of cases—almost constantly in the last few weeks, but sometimes in the whole course of the disease—the urine is more or less scanty, sometimes with intervals of a few days in which it is normal or increased in quantity. No doubt these discrepancies of opinion depend mainly upon the degree of care taken by different observers to recognise an underlying lardaceous change in kidneys that would formerly have been set down as examples of parenchymatous nephritis. Wagner, for instance, says that in the post-mortem room the lardaceous form of Bright's disease is seen more frequently than any other—"*much* more frequently than chronic parenchymatous nephritis or cirrhosis of the kidney." And I must confess that my experience at Guy's accords very much with his. So that, when Dr Grainger Stewart states that he has seen a considerable number of cases in which the lardaceous change was uncomplicated, and in which polyuria existed from first to last, I cannot help thinking that these after all formed an insignificant minority of the cases of lardaceous disease which he must have had under his observation, but of which he may possibly have overlooked some. The specific gravity of course varies with the quantity; it may be as low as 1003 or 1005, or it may reach 1025.

In all but exceptional cases *albumen* is present. Lecorché has maintained that so long as the lardaceous change is uncomplicated with nephritis the urine is non-albuminous. But Bartels denies that this is correct; and one at least of our cases at Guy's Hospital affords proof to the contrary. On the other hand, there can be no doubt that Bartels himself goes too far when he declares that albuminuria is always present, except perhaps at the very commencement of the affection, when the change in the vessels is just beginning. Many observers have noted that even in cases in which albumen is sometimes to be detected, it may at other times be absent from the urine. And Cohnheim speaks of having made autopsies in several cases in which albuminuria was said to have been altogether wanting. The quantity of albumen varies greatly. There may at first be only a little; but the rule is that the quantity is large, and it may reach from 1 to 3 per cent. Whether or not the presence of such considerable amounts of albumen may be taken as evidence that there is nephritis as well as the lardaceous change seems as yet to be doubtful.

In some exceptional cases there may be found in the urine glistening epithelial cells which turn reddish brown with iodine, from having undergone the lardaceous change before being shed. The discovery of them is probably conclusive as to the original nature of the renal affection. The urine is generally pale and transparent, throwing down no deposit, or at most a few hyaline casts and fatty epithelial cells. Blood is very rarely present, even in small amount. The statement made by Senator some years ago that the presence of paraglobulin in large quantity is characterstiic of this rather than of other forms of Bright's disease, has not, I believe, been confirmed since the introduction of the most recent method of separating paraglobulin from serum-albumin (p. 434). On the other hand, when there is much nephritis the urine may be high coloured, and may give an abundant precipitate containing lithates, as well as numerous hyaline and granular casts. Bartels mentions a case in which the average daily secretion amounted to less than seventeen ounces. And for several days before death the urine is often scanty, and sometimes altogether suppressed.

The other symptoms which accompany lardaceous disease of the kidneys are very variable and uncertain. It must not be supposed that cachexia and wasting are necessarily present. Bartels speaks of having had patients who were able to follow laborious occupations at a time when there was unequivocal evidence of the existence of this renal affection. And one of my cases at Guy's Hospital is that of a man, aged thirty-six, who was admitted with a fractured spine, the result of a fall while he was carrying a sack of barley on his shoulders; lardaceous changes were found in the liver, the spleen, and the adrenal bodies, as well as in the kidneys, which were enlarged, and weighed sixteen ounces.

Dr Stewart lays stress upon a pasty or waxy complexion, with a little pigment about the eyelids, and distension of small blood-vessels upon the cheeks, as being suggestive of this form of renal disease. He appears to me to make far too little of *dropsy* as a symptom of lardaceous disease of the kidney. It is no doubt true that dropsy is often wanting, especially when there is diarrhœa from a coincident affection of the intestinal mucous membrane. But I am certain that this symptom is frequently present in an extreme degree, affecting the face and the arms and the whole body, exactly as in cases of the "large white kidney" for which (as we have seen) cases really lardaceous have so often been mistaken, both at the bedside and after death. Bartels declares that when dropsy does occur it is ordinarily confined to the lower limbs and to the abdomen. But though I find notes of some few cases which, taken by themselves, might be considered to support this statement, I cannot endorse it as being generally applicable. When such a limitation of the dropsy is observed, I should myself regard it as an evidence that the immediate cause of the effusion was failure of the heart's action, on the grounds stated at p. 442. Now, it has been noticed by all recent writers on the subject that the heart is seldom found enlarged in cases of lardaceous disease of the kidneys. Cohnheim asserts that even when there is wasting and shrinking of the kidneys the heart invariably remains small unless the lardaceous change in them was preceded by the cirrhotic. But I find notes of four cases, in each of which the heart was found after death to weigh from eleven to thirteen ounces; and in two of these the kidneys also were enlarged, weighing fourteen ounces and eighteen ounces respectively. The frequent absence of cardiac hypertrophy seems to me to be sufficiently explained by the cachetic and anæmic condition of the immense majority of those whose organs present the lardaceous change; and I do not see that it proves (what is very unlikely) that diffused nephritis affecting lardaceous kidneys differs from that which arises under all other circumstances in having no effect upon the heart. But if this view is correct, and if there is really some degree of increase of arterial tension, there is certainly nothing to be wondered at in the occurrence of cardiac dropsy, as a result of the failure of the heart to respond to the increased calls upon it.

Again, both *retinitis* and *uræmia* are said to be rarely observed in cases of lardaceous kidney affection. But I find notes of three cases at Guy's in which death was ushered in by convulsive seizures. One patient, who had been in the hospital nine months previously with dropsy, was readmitted four days before death in a state of collapse, with cold and blue extremities, the result of severe diarrhœa and vomiting. In several instances acute peritonitis, or acute pericarditis, was what immediately brought the disease to a fatal termination.

Diagnosis.—From what has been stated with regard to the clinical effects of the lardaceous affection of the kidneys it is, I think, evident that, apart from other circumstances, neither the characters of the urine nor any other symptoms can be relied on to suggest its real nature, as distinguished from the other forms of Bright's disease. What, in fact, generally enables a diagnosis to be made is either (1) that indications of lardaceous change are presented by the liver, or by the spleen, or by both of them; or (2) that one or other of the known causes of this change is traceable. As to the first of these points I need not recapitulate what is stated elsewhere (p. 311) in reference to the physical characters of enlargements of the liver dependent upon a lardaceous change. Diffused lardaceous infiltration of the spleen and lardaceous disease of its Malpighian follicles forming the "sago-spleen" may both be well marked without the bulk of the organ being increased. It must therefore be borne in mind that the spleen, at any rate, does not always become increased in size. Further, even when both spleen and liver are considerably enlarged, there may be no possibility of detecting the enlargement in patients who are fat or flatulent or dropsical. Knowledge of the causes of the affection is therefore all-important.

Ætiology.—Most writers put the causes of the lardaceous change in a somewhat different way from that in which I should be disposed to arrange them. Bartels, for example, speaks of scrofula, chronic tuberculosis, and syphilis, as being all alike constitutional diseases which predispose to the development of this affection, probably in consequence of their liability to induce chronic ulcerations of bone, skin, or mucous membrane. He also admits, however, that it may show itself in any individual as the result of chronic and protracted suppuration. Now, it appears to me that such a method of statement is incorrect in two important respects. In the first place, it is certain that *syphilis* in some mysterious way induces the lardaceous change in cases in which pus formation has occurred only to the most insignificant extent, if at all. In the second place, it is altogether doubtful—and to clear up the doubt would be very difficult, if not impossible—whether scrofula or chronic *tuberculosis* has any influence in the same direction, apart from its tendency to set up ulcerative and suppurative lesions. It therefore seems as though we might reduce the known causes of the lardaceous change to two—syphilis and chronic *suppuration*—which may of course be combined, but either of which is capable of acting separately. Some pathologists mention, as an additional cause, the cachexia caused by ague, but I have not met with any recorded observations which seemed to me to establish its claim to be so regarded, and in which the possibility that the patients might also be syphilitic appeared to have been duly taken into consideration. It is, however, by no means improbable that there may be other causes besides the two above enumerated; as I have remarked at p. 339, there is some reason for thinking that the lardaceous change occurs in association with Hodgkin's disease without there having been sufficient suppuration to account for it.

In 1876 I brought before the Pathological Society a tabulated statement as to what appeared to have been the ætiology of 244 cases of lardaceous disease of the viscera, collected for me by Mr Lancaster from the post-mortem records of Guy's Hospital over a period of twenty-one years. In 154 there had been prolonged suppuration; 67 of these were cases of phthisis; in 51 there was disease of some joint (generally the hip- or knee-joint) or caries of the spine, or of some other bone; in the remaining 36 there were a variety of affections, amongst which I may mention empyema, dysentery, calculous and scrofulous pyelitis, ulcerating, cancerous, or sarcomatous tumours, cystitis from stricture, and bedsores of long standing as the result of disease of the spine. There were also five other cases in which there had, indeed, been pus formation, but in which it seemed doubtful whether this had been enough to afford a reasonable explanation of the occurrence of the lardaceous change. I may cite one instance in which there had been chronic discharge from one ear as well as from the nose; another, in which one testicle had been inflamed and suppurating as the result of a blow two months and a half before death, but with open discharge only for a fortnight; and a third, in which there was merely tubercular peritonitis with caseous disease of the mesenteric glands. In five or six of the 154 cases that appeared to be clearly due to suppuration, this had had a definite starting-point, so that some idea could be formed of the length of time required for the development of lardaceous lesions. One patient had had a carbuncle eight months; another had had pelvic cellulitis for exactly the same period; a third had had a bedsore seven months; a fourth was affected with a sarcomatous growth, which had been discharging for four months. In a fifth case there had been fracture of the spine three months before death, bedsore two months and a half, and also a double empyema; but syphilis was probably also present, inasmuch as the testes presented fibroid changes and there was a scar in the groin. A sixth patient had had his leg amputated three months and a half before death, on account of a compound fracture with abscess; in that instance it is particularly noticed that both in the liver and in the spleen the lardaceous change was just beginning.

On the other hand, among the 244 cases of lardaceous disease there were 76 in which there was satisfactory proof (either from history, or from post-mortem appearances, or from both together) of syphilis; and in three others there was at least a suspicion of its presence. In about 34 of the 76 cases there was evidence of there having been bone disease or suppuration, leaving 42 to be ascribed to syphilis *per se*. In no instance is it stated that the syphilis had been inherited; but Bartels speaks positively of having seen lardaceous affections in cases of inherited syphilis attended with ulceration of skin and bone.

Of the 244 cases, there are thus left only six examples of lardaceous disease that were not accounted for by the presence either of syphilis or of suppuration. And in some of them the notes of the autopsy are incomplete; the state of the testes, in particular, being unrecorded. Now, it seems to me far from unlikely that in each of these six cases the cause of the lardaceous change was really syphilis. When we consider how slight and accidental are very often the lesions on which we rely as proofs of the presence of syphilis in the dead body—and also how utterly inadequate they are in themselves to produce either cachexia or the lardaceous change—it cannot but appear almost certain that there must be other cases in which no syphilitic lesions whatever are discoverable, but in which syphilis is nevertheless really present. Six cases among 244 do not seem to me at all too numerous to bear this explanation; and I should be ready to add to them some of the five other cases in which, although suppuration had occurred, there was a doubt as to its being adequate to the production of the lardaceous change.

Practical bearing of these facts.—In regard to the *prevention* of lardaceous affections, it clearly is of great importance that all inflammatory diseases likely to lead to protracted suppuration should be so treated as to shorten their course as much as may be. And experience may perhaps hereafter show that in cases of syphilis it is desirable, on this account, as well as with the direct object of eradicating the obvious symptoms of the disease, to continue the administration of mercury and of iodide of potassium longer than has hitherto been usual.

It must not, however, be supposed that in all the cases which after an autopsy are positively set down as dependent upon syphilis the presence of this disease has been, or even could have been, recognised during life. On the contrary, there are a great many instances in which it is only by the discovery in the dead body of such lesions as fibroid degeneration of the testes, or small gummata or cicatrices in the liver, that the syphilitic character of the case is made out. In this respect there is a wide difference between the two great causes of the lardaceous change. For the occurrence of suppuration to an extent adequate to produce lardaceous affections can hardly ever be overlooked, except perhaps when it takes place from the intestinal canal (as in dysentery) or in those exceptional cases in which there is no external discharge of pus at all, but merely a large deep-seated abscess, the contents of which undergo slow inspissation and caseation.

It may possibly be thought that the clinical diagnosis of lardaceous disease of the kidneys from other forms of Bright's disease is, after all, a matter of no great moment, since it is to the diffused nephritis which is present in both sets of cases that most of the symptoms are really due. But there is some reason to believe that, both as regards prognosis and with a view to treatment, it is important not to overlook the possible presence of the lardaceous change. When general dropsy sets in, the downward progress of cases with lardaceous kidneys is often more rapid than that of other forms of Bright's disease. But, on the other hand, it not infrequently happens that albumen is detected in the urine of syphilitic patients several years before serious symptoms appear. There is also satisfactory evidence that such cases, even when dropsy occurs, sometimes end in recovery, the urine gradually resuming its normal characters. Two instances of this are recorded by Dr Dickinson in vol. xxx of the 'Pathological Society's Transactions.' One of his cases is that of a gentleman who contracted syphilis in 1861, and who in 1874 began to suffer severely from periosteal nodes, and soon afterwards was found to have his legs shapeless with œdema, his urine being also loaded with albumen, so that the coagulum amounted to one fourth or one half of the fluid in which it was formed. Under systematic treatment, assisted by a residence on the Riviera during four winters, the disease slowly subsided, and by June, 1879, the urine was quite natural and the patient himself apparently in a state of perfect health. As to what changes take place in the kidneys when recovery occurs nothing is yet known. It seems not impossible that the glomeruli and other vessels that were lardaceous may return to a normal condition, at any rate in cases in which the albuminuria is only of short duration. But in protracted cases, such as Dr Dickinson's, it is far more likely that the affected parts of the kidneys shrink and undergo atrophy, and that parts that had escaped the disease take upon themselves the whole renal function, possibly undergoing hypertrophy to enable them to perform it.

In practice I fear that the only way in which one can escape the danger of overlooking among one's cases of Bright's disease some at least of those that are lardaceous, is to give the iodide of potassium freely to many patients in whom the cause of their renal affection is altogether obscure, especially if there is any reason to suspect dissipated habits at an earlier age.

The period of life at which lardaceous lesions due to syphilis are most apt to occur is from thirty-one to forty. I find that at Guy's Hospital there have been fourteen fatal cases in persons between those years against six in persons between twenty-one and thirty. As the result of protracted suppuration, on the other hand, the affection has been slightly more frequent during the earlier period; it occurs, too, in adolescents, and even in children; there has been one case at Guy's Hospital in a boy only four years old. Above the age of fifty lardaceous disease is decidedly uncommon, but we have had one instance of it in a man of sixty-five. Among the cases that I have collected male patients have been more numerous than females in the proportion of two to one.

CIRRHOSIS OF THE KIDNEY. *Red granular atrophy of the kidney. Chronic interstitial nephritis.*—As I have already remarked at p. 430, I adhere to what may be termed the English view with regard to granular atrophy of the kidney, believing that those cases in which this affection is secondary to a parenchymatous nephritis or to a lardaceous change in the vessels should be distinguished absolutely from those in which it constitutes a primary morbid process. One proof of the reality of this distinction seems to me to be afforded by the difference in the appearance of granular kidneys at different periods of life. In children, and even in adults under the age of twenty, twenty-five, or perhaps thirty years, I am not aware that the red or brown granular kidneys are ever seen which are so frequent in middle-aged and in old people. So far as I have observed, the granular kidneys of early life are always full of opaque whitish yellow spots or patches. It is true, doubtless, that these tend to become fewer and less conspicuous as the affection becomes more and more advanced; and I can quite conceive the possibility that in some exceptional instances they may altogether disappear, leaving the organ in a condition exactly like that which is met with at a later age. But even if the means of distinguishing them should thus sometimes fail it would not at all follow that the two diseases should not still be held to be different on the strength of the obvious differences presented by the large majority of cases.

On the other hand, in older patients I freely admit that the criterion afforded by the appearance of the kidneys not infrequently ceases to be applicable. The reason is that when a part of the renal substance has been destroyed by cirrhosis the remainder is apt to become affected with the parenchymatous change. Thus, I have found in the records of post-mortem examinations at Guy's Hospital no fewer than thirteen cases in which the kidneys of patients who had been the subjects of gout, and whose joints contained urate of soda, were wasted and granular, but at the same time whitish yellow in colour. Of most of these cases the clinical history given in the post-mortem notes is too imperfect to enable one to determine satisfactorily the period at which the second affection supervened. But in 1873, a woman, aged forty-four, a gin-drinker, died after an illness of seven weeks' duration, which she said began one day with pains in the loins while she was working in a cold washhouse. On the following day the face was swollen, vomiting then set in, and afterwards the legs and the abdomen became swollen. The urine was albuminous, of sp. gr. 1012. On post-mortem examination the kidneys were found mottled with yellow, but they weighed only eight ounces, and were granular on the surface, and their cortex was much narrowed. The arteries were rigid. The heart weighed twelve ounces, the left ventricle being hypertrophied.

The pathologist, no doubt, is altogether unable to distinguish such "mixed" cases as these from cases of primary parenchymatous nephritis in an advanced or granular stage if he has no clinical history to guide him, and if none of the joints contain deposits of urate of soda, indicative of a gouty origin for the disease. But in this country the "mixed" cases after all form an insignificant minority in comparison with the very numerous cases in which the kidneys are purely cirrhotic. Of these last I have before me notes of considerably more than one hundred. What proportion of them were associated with gouty deposits in the joints I cannot say, for unfortunately the joints are still too often forgotten in our post-mortem examinations. But we have very often found the joints affected in the bodies of those who were not known to have had any gouty attack. At the meeting of the International Congress in 1881 Dr Ord stated the results of some investigations which had been made at St Thomas's Hospital, and from which it appeared (if I understand them correctly) that among some twenty-four or twenty-five cases of granular disease of the kidneys there were sixteen in which gouty deposits were present in the joints, eight or nine in which they were absent. It is not unlikely that the rarity of gout in Germany brings with it a corresponding rarity of renal cirrhosis, and this may be the chief reason why German writers fail to recognise the latter affection as distinct from other forms of Bright's disease. But I find that they speak of a "senile atrophy," which they regard as devoid of clinical importance. And probably under that name they put aside many of the less marked instances, especially when the cause of death is not obviously dependent upon the state of the kidneys. I am not aware that in this country any pathologist admits that in old people the kidneys are naturally liable to any change unless it be a slight shrinking, corresponding with the loss of weight in the tissues generally that comes with advancing years.

Ætiology.—As regards the *period of life* at which death occurs in persons with cirrhotic kidneys I find that in eighteen cases at Guy's Hospital it was between 31 and 40 years, in thirty-nine between 41 and 50, in thirty-six between 51 and 60, in twenty-four between 61 and 70, in four between 71 and 80. Below the age of 30 I am not sure whether any typical instances have been met with, but several of the persons who were between 31 and 40 had had gout, and presented the disease in a perfectly characteristic form. My figures correspond pretty closely with those given by Dr Dickinson. They are, of course, very unlike those of E. Wagner, who mixes up together all forms of "contracted kidney." The proportion of males to females in my cases was about exactly as two to one.

Next to *gout,* the best known cause of cirrhosis of the kidneys is chronic poisoning by *lead.* The disease is accordingly seen in painters, in printers, and in typefounders, and sometimes in those whose tissues have accidentally become impregnated with the metal, or by using snuff wrapped in foil containing it. But lead-poisoning also frequently produces gout; and I have not been able to learn whether the renal affection is often, or indeed ever, found in the bodies of those whose joints are entirely free from deposits of urate of soda. Most observers think that excessive indulgence in *alcohol,* altogether apart from its tendency to set up gout, is a common cause of granular atrophy of the kidneys. And although Dr Dickinson disputes this, I should for my own part be strongly disposed to agree with them. But the reports of post-mortem examinations at Guy's Hospital do not bear out the statement sometimes made that the renal affection is frequently found in association with cirrhosis of the liver, in spite of the apparent resemblance between the two diseases from a histological point of view. ('Guy's Hosp. Rep.,' 3rd series, vol. xx, p. 196).

Anatomy.—In its earlier stages, renal cirrhosis is by no means a very conspicuous morbid change. The kidneys may be of natural size and colour; and beyond the fact that the normal arrangement of the different sets of tubes is no longer visible upon the cut surface of the cortex, and that the capsule is thick and too adherent, there may be nothing to suggest the presence of any disease. One is sometimes surprised to find with the microscope to what an extent the cortex has been destroyed. In advanced stages the case is very different. The capsule is greatly thickened and cannot be stripped off without the cortex tearing and coming away with it. The surface of the kidney is covered with little projections or granulations. These, in uncomplicated cases, are of a red or reddish-grey colour. They no doubt consist (like the granules of a cirrhotic liver) of portions of the cortex which are less altered and wasted than the rest. Charcot maintains that they correspond in position with the summits of the "medullary rays," or columns of straight tubes that traverse the cortex, the depressions between them answering to the intervening tracts of convoluted tubes. However, Weigert and E. Wagner dispute the correctness of this opinion. The reduction in the thickness of the superficial parts of the cortex is often extreme. The bases of the pyramids may be separated from the exterior of the kidney, by a layer of tissue only a line or so thick. The inter-pyramidal portions of the cortex are not generally wasted to the same degree. As I have already stated, I believe that in the early stages of the morbid process they undergo a compensatory hypertrophy. The weight of the two kidneys is often reduced from eight or nine ounces to five, four, three, and sometimes even to two and a half ounces. The smallest kidneys that I have ever heard of were a pair that were found by Dr Wilks to weigh thirty grains short of an ounce and a half. However, neither the diminution in size, nor the loss of weight, gives a fair idea of the destruction of the renal cortex. For the pelvis is proportionately wider than in the healthy organ, and is filled with what appears an excessive amount of adipose tissue, over which the substance of the kidney is spread out as a thin shell.

Histologically, the tubular structure of the renal cortex is found to be replaced by connective tissue in various stages of development. At first there is merely a "small-cell infiltration," which forms foci scattered here and there through the organ, especially round the capsules of the glomeruli, but with prolongations between the tubes immediately adjacent to them. Ultimately the small-cell infiltration undergoes conversion into fibrous tissue, in which there are generally very few blood-vessels, though these are sometimes remarkably wide. Embedded in this fibrous tissue there are usually to be seen more or less numerous relics of tubes. These are mostly narrow and their channels are filled with cells very unlike those of the normal renal epithelium, or with hyaline casts. But sometimes, in extensive tracts, hardly a trace of renal structure can be detected. Contrasting with them are to be seen areas in which the tubes are comparatively unaltered, except that they are generally wide and patent, with flattened epithelium.

The glomeruli and Malpighian capsules are more or less completely destroyed in the fibrous tracts of a cirrhotic kidney. The capsules often become thickened by the formation of concentric layers, giving them a fibrillated appearance. The tufts degenerate into a structureless material, containing only a few scattered nuclei. Ultimately nothing may be left but a round translucent mass, of which the diameter is not more than one half or one third of that of the normal glomerulus. By the shrinking of the intervening tissue these "glassy globes" are often drawn close together, so that a number of them are seen in the same microscopic field. On the other hand, in those parts of the kidney which still retain a tubular structure the glomeruli may be considerably larger than natural.

It is held by some pathologists that degeneration of the glomeruli is the primary change in the tissue of the kidney in this disease, being itself perhaps dependent upon an *arteritis obliterans* affecting the smaller branches of the renal artery in the cortex, and even the afferent vessels of the tufts. The thickening of even the larger arterial twigs, indeed, constitutes one of the most obvious morbid appearances in a red granular kidney; they stand out upon the cut surface of the organ with patent mouths, like so many little quills. Thoma, however, has recently shown ('Virchow's Archiv,' 1877) that when a glomerulus becomes obsolete, its afferent artery may remain pervious, and may open straight into the efferent vessel or into the interlobular capillary network.

In many cases the histological characters of a cirrhotic kidney are further complicated by the presence of innumerable minute *cysts*; certain observers even describe cases of this kind under a separate name as "micro-cystic." The immense majority of them are invisible by the naked eye, but some may be of all sizes up to that of a millet-seed, or even of a pea. They may make up by far the larger part of a microscopical section. Their contents are often a yellowish-brown jelly-like substance, which can be turned out of the larger cysts as a solid mass. With regard to their nature there was at one time some controversy. Mr Simon suggested that they might be formed out of overgrown epithelial cells; others have thought that they arose out of Malpighian capsules. But the former mode of origin seems very improbable, nor can any analogy be cited in support of it; the latter would not account for more than a fractional part of the numberless cysts that are often present. Accordingly, all pathologists are now agreed that they consist of portions of tubes that have become cut off, and have assumed a spherical shape. Not only are they sometimes arranged in rows, like beads in a necklace, but intermediate forms are often met with—cylindrical cavities, with partial constrictions here and there in their course.

Another appearance that is observed in cirrhotic kidneys is due to the deposition of *lithic acid* or of its salts in the renal tissues. It consists in the presence of minute whitish-yellow grains scattered through the cortex, or arranged in lines in the pyramids, the summits of which may even appear to be completely encrusted by them. Some of them are amorphous, some are made up of bundles of acicular crystals. They appear to lie partly between the tubes, partly in their interior. Dr Garrod seems to think that such deposits are the results of gout, and that, acting as foreign bodies, they produce albuminuria and set up the kidney disease in association with which they are found after death. But I believe that they are often found in cases in which there is no other evidence of gout; and they are commonly seen in Germany, where gout is rare.

Symptoms.—In the slighter degrees, or in the earlier stages, of cirrhosis of the kidneys the urine may be normal in every respect. Dr Grainger Stewart, for instance, relates a case in a man of sixty-five, who died of phthisis, and who passed forty ounces daily, the specific gravity being 1020. But when the disease is advanced, the *quantity of urine* is often greatly increased and its specific gravity is very low. The quantity may amount from 70 to 210 ounces daily; in a case recorded by Bartels it was measured on a single occasion from 8 p.m. to 8 a.m. only, and was found during that period to reach 210 ounces. It is commonly secreted more abundantly at night than in the day. Bartels had the urine of one of his patients measured for twenty-six days, that passed from 10 p.m. to 7 a.m. being separated from that passed from 7 a.m. to 10 p.m.; during the nine hours of night the average quantity was found to be seventy-seven ounces; during the fifteen hours of day only forty-eight ounces. The *specific gravity* varies from 1004 to 1010. The urine is faintly acid, pale, clear, depositing no sediment, or only a very slight cloud. It usually contains only a very small quantity of *albumen,* less than ·5 per cent; the whole amount of albumen in the twenty-four hours is said by E. Wagner to be not more than from twenty to ninety grains. Indeed, unless care be used in applying the tests, the presence of albumen may be altogether overlooked, even when the urine really contains it; after pouring nitric acid through the urine one must not forget to allow time for the opalescent zone to appear. But it not seldom happens that albumen is entirely absent for days or weeks together. Or the urine passed during the night may be constantly free from it, while it is as regularly present in that passed during the day. Whether the urine is ever absolutely non-albuminous throughout the whole course of the disease seems to be still open to question. There have been cases in which no albumen has ever been detected, but the doubt is whether the urine has been tested sufficiently often. When albumen is more or less constantly present it is often more abundant in the day urine than in the night urine; and this is the case even when the patient is kept always in bed. *Casts* are often entirely absent; when any are found they are commonly hyaline and narrow, but sometimes opaque and granular. The amount of *urea* excreted daily seems, according to recent observations, to be not so deficient as used to be supposed. It is true that the proportion in a given quantity of urine is generally not more than 1 or 2 per cent., but so abundant is the urine itself that Bartels found in four of his cases that the daily average of urea ranged from 296 to 522 grains.

Gouty persons, who have been accustomed to pass high-coloured urine, becoming thick with urates, are often deluded by the idea that their kidneys are performing their functions much better than before, when the secretion becomes pale and clear as the result of the development of cirrhotic changes in them. But in cases in which the flow is much increased, and in which there is consequently great thirst, it frequently happens that patients take alarm and seek advice for what they imagine to be diabetes. When the urine is tested and no sugar is found in it, this supposition is of course negatived. But, as I have observed at pp. 345–6, it sometimes happens that if albumen happens for a time to be absent, the diagnosis of *diabetes insipidus* is wrongly given. Even when the quantity of urine passed is not noticed to be excessive, the patient may be obliged to get up out of bed three or four times every night to micturate. He may not complain of this, and one has to learn whether it is so by asking him. And if nocturnal irritability of the bladder has existed for a considerable time, it may be fairly inferred that the renal affection is of at least as long standing.

The character of the *pulse*, and the state of the *heart* form very important elements in the diagnosis of cirrhosis of the kidney, especially when no albumen is found in the urine. I have already fully discussed them at p. 447 *et seq.*

Clinical forms.—The disease comes under observation in several different ways:

1. Some patients only complain of *weakness and exhaustion;* and the most conspicuous physical change discoverable in them is that they are anæmic and wasted, with flabby muscles. I remember a case some years ago that was set down during life as one of idiopathic ("pernicious") anæmia. The patient was a man, aged fifty-nine.

2. In certain patients the chief symptoms are *gastric or intestinal;* uncontrollable vomiting, or diarrhœa, or both together. I think it is especially in such cases that the breath is sometimes horribly fœtid, as mentioned by Bartels.

3. In a good many instances what brings the patient under medical observation is the occurrence of *acute pneumonia, pleurisy,* or *pericarditis.* Peritonitis, which is common in parenchymatous nephritis, I have never seen as a complication of renal cirrhosis. The acute thoracic inflammations are often rapidly fatal, and I think that the common belief is that pericarditis, when it occurs in a case of kidney disease, is almost always attended with grave symptoms, and dangerous. But I have sometimes known it to give rise to but little disturbance of the general health, and to subside after a while more or less completely. And even when it is still active at the time of the patient's death, the amount of lymph exuded is often but small, so that I have been by no means clear whether it has played an important part in bringing about the fatal issue.

4. *Cerebral hæmorrhage* is very often the cause of death in renal cirrhosis, and in many cases it is not preceded by any symptoms that are recognised as indicating that the patient is seriously out of health, or affected with any organic disease. Sometimes the occurrence of hemiplegia is due not to extravasation of blood, but to softening of a part of the brain-substance, as the result of arterial changes.

5. I do not find that *uræmia* is of very frequent occurrence in renal cirrhosis. Among 120 cases at Guy's Hospital there seems to have been only fifteen or sixteen that ended so. Three of them occurred in patients between thirty-one and forty, six between forty-one and fifty, two between fifty-one and sixty, two between sixty-one and seventy, two between seventy-one and eighty. The statement commonly made that this form of kidney disease is the one above all others in which uræmia is apt to occur, appears

to be traceable to the fact that the advanced stages of parenchymatous nephritis have been so generally mixed up with it. Headache, giddiness, and other cerebral symptoms are often complained of; but such symptoms appear in many cases to be dependent upon the diseased state of the intracranial arteries rather than upon the condition of the blood. In 1880 a man, aged forty, was admitted into Guy's Hospital for pain and heat of the head. He was found to have albuminuric retinitis; afterwards he became melancholic with suicidal tendencies. A point of considerable importance is, that in patients affected with this form of Bright's disease (or, I suppose, with any other form in which there is extensive destruction of the renal cortex) the administration of even a small dose of opium or morphia is followed by fatal cerebral symptoms. Thus, I have notes of one case in which a grain of opium, prescribed for pain in the head, appeared to be the cause of convulsions and stupor that ended in death; and in another case like results seemed to be due to the administration of a third of a grain of morphia for lead colic.*

6. Sometimes the patient comes under observation with renal *dropsy,* having the characters described at p. 442. I believe that such cases are always examples of the supervention of a parenchymatous nephritis upon an antecedent cirrhosis and that at the autopsy the renal affection is found to be of the "mixed" kind. But unless the patient has been under observation previously it may be impossible to diagnose the presence of any but recent changes in the kidneys. The state of the urine is indistinguishable from that which might be produced by them alone. In all persons beyond middle age affected with renal dropsy it is very important, with a view to prognosis, that the probability of there being chronic as well as acute lesions should be borne in mind.

7. But by far the most important group of cases of cirrhosis of the kidneys is that in which the main symptoms are *cardiac,* the patient coming under observation with dyspnœa, palpitation, and dropsy of the dependent parts of the body. Dr Mahomed ('Guy's Hosp. Rep.,' 1879) stated that such cases constitute 17 per cent. of all those in which this affection of the kidneys is found after death, and my own analysis of a somewhat larger number of cases than his yields an even higher figure. After death the left ventricle is usually found dilated as well as hypertrophied, and its walls may be considered to have yielded to the excessive strain upon them. But sometimes the post-mortem evidence of dilatation is by no means complete. Degeneration of the muscular tissue of the heart is probably in many cases the cause of its failure. Its substance may be soft and flabby, and under the microscope the fibres may be seen to be granular and to have lost their consistency, breaking into short fragments when an attempt is made to separate them with needles. A fibroid change in the papillary muscles of the mitral valve is not uncommonly seen; they taper gradually into the tendinous cords, instead of appearing as stout fleshy columns. Whether the mitral valve often leaks is, I think, doubtful. A systolic apex-murmur, having some of the characters of the mitral regurgitant murmur, may be heard in many cases, but I have been disposed to look upon dilatation of the left ventricle, without valvular lesion, as an adequate cause for such a murmur. It is not often that positive proof of regurgitation is afforded at the autopsy by the presence of "ripple lines" on the posterior wall of the left auricle, as in a case under my care in 1878—79, recorded by Dr Mahomed ('Guy's Hosp. Rep.,' 1879). Sometimes I have observed that

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* [While these sheets were passing through the press, a patient of mine suffering from cancer of the throat died comatose from subcutaneous injection of a fourth of a grain of morphia. The kidneys were found to be the seat of chronic cirrhosis with wasting of the cortex.—Ed.]

the extremity of the anterior flap of the mitral valve was thickened, and that it could be readily bent inwards while the chordæ were stretched or that it had a small vegetation upon it. Whether these appearances afford evidence of regurgitation I am not sure. The occurrence of actual endocarditis secondary to Bright's disease is not, I think, common. But, undoubtedly, cases are every now and then seen which appear to admit of no other explanation.

Until recently cases of heart failure secondary to cirrhosis of the kidneys were almost always regarded during life as examples of a primary morbis cordis. Over and over again, when the autopsy showed that the renal changes had reached an extreme point, have I found that the diagnosis sent down from the wards was "mitral regurgitation." But I must not omit to add that sometimes one may be fairly in doubt as to the correct interpretation of the post-mortem appearances. The kidneys, perhaps, are of fair size, although hard, red, and glistening. Even the microscope, while revealing a certain amount of fibroid change, with degeneration of some glomeruli, may leave one in doubt whether this is more than an accidental feature of the case, especially if the patient was advanced in years. Dr Mahomed, too, points out that mitral stenosis and other effects of rheumatic inflammation of the heart are not very rarely found in association with cirrhosis of the kidneys, though I suppose that the relation between the two affections is only one of coincidence. In the clinical diagnosis of the cardiac dropsy secondary to renal disease from that which is primary the state of the urine appears to be of little assistance. In either case it may be scanty, high coloured, and loaded with urates, and may contain more or less albumen as well as casts. E. Wagner says, however, that in cases of renal cirrhosis, even when the urine is scanty, its specific gravity is seldom above 1012; and he cites an observation of Traube's that in extreme instances it may remain pale and of low specific gravity even when there is great obstruction to the venous circulation, or (on the other hand) when some febrile disease, such as acute pneumonia, develops itself. But I have notes of two cases in each of which the specific gravity of the urine ranged from 1020 to 1025, although at the autopsy the kidneys weighed only seven and a half or eight ounces, and were very granular. And in a footnote to Dr Southey's translation of Bartel's work on 'Bright's Disease' a case is mentioned in which in spite of very great wasting of the kidneys the urine had a specific gravity of 1028, and deposited urates freely on cooling. Indeed, whatever may be the rule as to the more advanced stages of the affection, I am sure that when the kidneys are still of good size, although the microscope may afterwards show that they have undergone extensive fibroid changes, they commonly yield a high-coloured secretion, of great density, during the time when cardiac dropsy is present. In all such cases it is to the pulse that we must look for guidance in our diagnosis. But I should doubt whether Dr Mahomed is right in laying stress upon a visible and tortuous condition of the temporal arteries, which, I think, often is merely due to local senile changes in the coats of these vessels. Again, as this observer himself admits, in cases in which there is much cyanosis from emphysema it is not safe to rely even upon a persistent pulse at the wrist as proof of renal disease. Probably the occurrence of high arterial tension in such circumstances is comparable with the rise of blood-pressure in the arteries that is observed at a certain period of asphyxia in experiments on animals (see vol. i, p. 10).

Consecutive Bright's Disease.—Under this name I propose to describe an affection of the kidneys which on account of its histological characters must be classed as a form of Bright's disease, but which is secondary to some morbid change in the renal pelvis, or in some part of the lower urinary passages. Cases of this kind have until recently attracted but little notice. The effects of pressure upon the kidneys have, indeed, long been recognised; but although it has been well known that when hydronephrosis occurs the pyramids are flattened and the cortex is thinned out, I think that the general idea has been that such appearances are only mechanical in their origin, and independent of any inflammatory process. On the other hand, both physicians and surgeons are, of course, familiar with suppurative nephritis, as the result of affections of the bladder or of the urethra. The cases to which I now desire to draw attention occupy an intermediate position. Like hydronephrosis, they are slow in their development; but, like suppurative nephritis, they are associated with very definite lesions of the renal tissue. A "consecutive interstitial nephritis" is, indeed, often associated with hydronephrosis, as I mentioned when I was discussing that disease; but it may occur when the pelvis of the kidney is perfectly healthy. In other cases it is cut short in its course by the supervention of a rapidly fatal suppurative process; but it is quite capable of destroying life without any such aid. By far the best account of it with which I am acquainted is that given by Mr Marcus Beck in the fifth volume of 'Reynolds' System' of Medicine.'

The appearances presented by the kidneys when affected with consecutive Bright's disease vary in different cases. There is an acute or subacute form in which they are swollen, mottled, with red and white patches, and of a soft consistence. But far more often they are very tough and hard, of a dull white or opaque waxy yellow colour. The surface is generally smooth; it is often very adherent to the capsule, which itself may be much thickened, and connected more firmly than is natural with indurated adipose tissue around it. But sometimes there is a marked granulation of the surface, so that the character of the affection may be exactly like that of ordinary cirrhosis of the kidneys. In 1879 I made an autopsy in the case of a boy, aged twelve, who had a hypertrophied and contracted bladder and great dilatation of the ureter and of the renal pelvis on each side; the two kidneys together, with much adventitious fat about them, weighed less than two ounces; the cortex was in most places shrunken to a thin red line; but, contrasting with the rest, there were some raised soft, pale yellow rounded nodules. Even when the cause of the disease is situated in the bladder, or in the urethra, the two kidneys are by no means necessarily affected to an equal extent. And when it is in the renal pelvis on one side only, that kidney may suffer alone. Thus, in 1876, in the case of a woman, aged forty-seven, who for twelve years had suffered from calculous pyelitis of the left kidney, I found the cortex of that kidney reduced to a thin shell of white fibrous material; the other one weighed nineteen ounces, and had undergone hypertrophy, though it was also affected with recent lardaceous and other changes which had evidently been the cause of the woman's death by uræmic coma. I have even met with an instance in which a calculus lay in one calyx of a kidney, and in which the corresponding part of the cortex was narrow and granular, all the rest of the organ being healthy. And when consecutive Bright's disease affects the whole of both kidneys, it often happens that the change is far more advanced in some parts than in others, so as to produce deep puckered cicatrices.

Histologically, the morbid process, as described by Mr Beck, is identical with that which characterises other forms of Bright's disease, in which the interstitial tissue is mainly affected. There is first an accumulation of immense numbers of leucocytes in different parts of the cortex, especially round the Malpighian capsules, but also between the tubes. The tubal epithelium is but little altered, being at most somewhat swollen and granular. Sometimes small extravasations of blood occur within the tubes, as well as into the intertubular tissue. The glomeruli become crowded with nuclei, and gradually shrivel into transparent bodies surrounded by thickened capsules. Ultimately the small-cell growth develops into fibrous tissue.

Consecutive Bright's disease occurs in various surgical affections of the urinary organs, such as stricture, disease of the prostate, villous disease of the bladder, stone in the bladder. But it is also seen by physicians in many different circumstances. Thus it may result from any of those diseases that are enumerated elsewhere as causes of hydronephrosis. It may also affect one kidney as a consequence of pyelitis; and in cases of tuberculous disease it often plays an important part in completing the work of destruction.

The clinical recognition of this form of kidney disease is beset with peculiar difficulties. The urine is often so altered as the consequence of cystitis or pyelitis that its characters lend scarcely any assistance. But if it happens to be free from blood and pus it may very probably contain neither albumen nor casts, though sometimes it is albuminous, and occasionally a few hyaline casts may be discovered in it. It is generally rather excessive in quantity, and of low specific gravity. But no conclusion must be drawn from the density of a single specimen, especially soon after surgical interference with the urinary passages. Mr Beck mentions a case of lithotrity in which the specific gravity of the first sample of urine passed was only 1003, whereas that of the whole twenty-four hours' urine was 1018. The quantity of urea excreted may be little, if at all, below normal; certainly it is often quite as great as can be expected if one takes into account the small amount of food which the patient can eat and digest, and the impairment of health that has been produced by the primary disease under which he is suffering.

Nor is the presence of consecutive Bright's disease clearly indicated by any marked general symptoms. In subacute cases there is often an evening rise of temperature to 100° or 101°, whereas the morning temperature may be regularly normal or even subnormal. The patient feels weak and languid, and steadily loses flesh. The tongue may be very foul, with a thick white or dirty fur, and may even become dry and brown. There may be much thirst, with little appetite for food, and more or less nausea. The skin is usually moist and clammy. There is neither tenderness nor pain in the loins. The patient's mental state is often placid; he may even be drowsy, like a person slightly under the influence of opium. Death sometimes occurs by exhaustion, sometimes by the supervention of some acute disease, sometimes by deepening stupor, or by uræmia. But if the primary disease can be relieved by surgical treatment, it is surprising how all the symptoms may subside that had appeared to indicate grave renal mischief, so that after all one may be left uncertain whether such consecutive mischief really existed and whether they were not merely dependent upon the primary disease itself. But even restoration to a fair state of health is no proof that the kidneys have not been damaged. And in the most chronic cases of all, in which the organs become yellowish white and tough, there are often for a long time no symptoms at all; the patient remains fairly nourished and is not anæmic; and he eats, drinks, and sleeps as usual.

It is a very important question whether this form of Bright's disease is liable, like the others, to produce cardio-vascular changes. Mr Beck says the heart does not become enlarged, although he admits that the renal arterioles show hypertropy of their muscular coats. E. Wagner, however, states that the heart was hypertrophied in each of five cases that came under his observation; in one instance there was a well-marked albuminuric retinitis, which indeed was the first symptom noticed by the patient. I have notes of two cases secondary to stricture of the urethra, in which the heart weighed sixteen and nineteen ounces respectively.

Cystic Disease of the Kidneys.—In discussing the anatomical characters of renal cirrhosis I have mentioned that in many cases there are found in the kidneys immense numbers of cysts, of all sizes up to that of a pea. Such kidneys are sometimes termed "micro-cystic."

There are, however, other cases in which the cysts are much larger, sometimes as big as oranges, and these are usually described separately under the name of "cystic disease of the kidneys." The organs then look as if they were each made up of a mass of rounded cavities, embedded in and separated by an abundant fibrous matrix. But in this there still exists remnants of secreting tissue, by which the renal functions have, however, imperfectly, been kept up. The pyramids, the calyces, and the pelvis are little, if at all altered; so that the affection must not be confounded with hydronephrosis. The cysts have walls of varying thickness. In the character of their contents they differ widely among themselves, even in the same kidney. Some are filled with yellow fluid, others with red, some with a gelatinous substance. The fluid always contains albumen, and sometimes blood-discs, leucocytes or plates of cholesterine; urea and uric acid are said to be generally absent.

There seems to be no doubt that the cysts are formed out of the tubes of the cortex, exactly like those which occur in cases of renal cirrhosis, and I altogether agree with the opinion expressed by Wilks and Moxon in their work on pathological anatomy, that the affection is a form of Bright's disease, notwithstanding one important clinical difference which exists between it and all the other forms. This is the fact that in cystic disease of the kidneys the organs are often so enlarged as to be felt during life through the abdominal walls. Bright many years ago recorded such a case, in which a distinct tumour was detected in the left loin some months before death, and afterwards another was discovered in the right loin. Dr Roberts relates a case in which he successfully diagnosed not only the renal nature of the two tumours that he discovered, but also the exact character of the disease by which the kidneys were affected; the tumours appeared to be soft, but not fluctuating, about as large as cocoa-nuts, but disproportionately long. After death one kidney weighed twenty-eight ounces, the other twenty-six. In many instances they have been much larger still. There occurred at Guy's Hospital in 1867 a case in which the right kidney weighed eighty-four ounces, the left fifty-three. In two other published cases the weight of the two kidneys together has been six and a half pounds, and eight and three quarter pounds respectively. But the most remarkable case of all is one which was brought before the Pathological Society by Dr Hare (vol. iii), in which the left kidney alone weighed sixteen pounds, the right being in a comparatively early stage of the disease, so that it was only of about twice the natural size. During life a tumour filled the whole left side of the abdomen.*

In all other respects the symptoms of cystic disease of the kidneys are like those of other forms of chronic Bright's disease. The urine is often rather excessive in quantity, and of low specific gravity. It seems generally to contain albumen, and in some cases hæmaturia has been observed from time to time. There has sometimes been marked emaciation with great prostration of strength. But many patients have not been known to be ill until they were attacked with uncontrollable vomiting, or with uræmic convulsions and coma, or (as has happened in two instances at Guy's Hospital) with cerebral hæmorrhage.

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* [Such monstrous cystic kidneys are sometimes congenital (Virchow, 'Ges. Abh.,' p. 864): to add to their interest, they are sometimes found associated with cystic disease of the liver; see 'Path. Trans.,' 1856, 1881, and 1883.—Ed.]

It is obvious that, apart from the presence of abdominal tumours, the clinical significance of this affection depends entirely upon the extent to which the renal cortex is destroyed. Sometimes even the portions of it between the cysts are more or less changed into fibrous tissue. On the other hand, a few scattered cysts, of various sizes, are frequently found after death in the bodies of those whose kidneys are in all other respects healthy; their presence is then unimportant.

Dr Roberts says that, among fifteen cases of cystic disease of the kidneys that he collected, ten occurred in men and five in women. Most of the patients were of middle age; the youngest was thirty.

Treatment of Bright's disease.—This must necessarily depend upon the stage at which the patient comes under observation and upon the symptoms which are then present.

Acute forms.—In the early period of parenchymatous nephritis, the patient should be kept strictly in bed. It is often wise to place him between blankets, and he should always wear a flannel gown with sleeves down to the wrists, so that the arms may not get cold when they are put outside the bedclothes. The diet should consist mainly of milk, but farinaceous foods may be allowed and also small quantities of beef tea or broth. When there is severe lumbar pain it may often be relieved by leeches or cupping glasses followed by hot poultices; but there seems to be no ground for supposing that local bloodletting is capable of affecting favourably the morbid process in the kidneys.

Some writers lay great stress upon keeping up an active flow of water through the glomeruli so as to wash out the tubes and empty them of the cell masses and casts by which they are blocked up. Thus, Dr Dickinson recommends his patients to drink water very freely, and Dr Grainger Stewart prescribes the *diuretic salts* of potass, or the spiritus ætheris nitrosi, and inhalations of oil of juniper.

I have been accustomed rather to order such medicines as are supposed to be *diaphoretic* in their action, especially the liquor ammoniæ acetatis (the use of which has been traditional at Guy's since the days of Addison), with or without small doses of antimonial wine.

Perhaps there is something in Dr Roberts's idea that the administration of the acetate or citrate of a fixed alkali such as potass is of benefit, because the salt is converted into a carbonate which tends to diminish the acidity of the urine and so to prevent its irritating the substance of the kidneys as it passes through them. There seems to be no objection at any stage of the disease to the use of such vegetable diuretics as broom, horseradish, and uva ursi, but not much good is to be looked for from them. Digitalis may be serviceable at all periods. Theoretically it might be supposed to have an injurious effect by augmenting arterial pressure, but this, Dr Mahomed says, is not found to be really the case to any appreciable extent. The importance of setting the skin freely in action is generally recognised. It is best effected by *baths*. One plan, advocated by Liebermeister, is that of placing the patient in a bath at about 100° and then gradually adding more and more hot water until it reaches 104° or 106°; he is left in the bath for half an hour or even an hour, and is afterwards closely packed in a sheet and warm blankets for two or three hours longer, during which time profuse sweating occurs. It is said that after such a bath the weight is often reduced by from two to four pounds, and even in children by half a pound or a pound. Ziemssen advises packing the patient in a sheet wrung out of hot water, and in one or two well-warmed blankets. In England we often employ "hot-air baths," a lamp being placed in the bed in which the patient lies, while the bedclothes are raised by means of a cradle so as to keep a confined space of air around his body. Any one of these methods may be repeated every day or every other day. Sometimes they cause faintness or headache, and a feeling of oppression, or a rise in the temperature of the body, and they have been known to bring on uræmic convulsions. It is especially in cases attended with severe dropsy that they require to be used frequently and energetically. Their effect upon this symptom is often very striking, and they may be followed by an increased secretion of urine as well as of urea. If there is prostration or great dyspnœa, the hot bath is sometimes dangerous. Of late years the pharmacopœia has been enriched with a powerful diaphoretic in pilocarpin, of which from one sixth to one third of a grain may be injected subcutaneously, or about twice that quantity taken by the mouth. It gives rise to a great flow of saliva and also to abundant sweating, but the latter effect is not always so well marked in persons who have Bright's disease as in those who are healthy. In some cases, however, it produces unpleasant symptoms, such as nausea, vomiting, or even collapse. E. Wagner says that these may be obviated by the administration of a little brandy, or wine, or coffee before the pilocarpin is injected.

The regular administration of *purgatives* is also of importance. The most usual practice is to give a dose of compound jalap powder twice or three times in the week. But if this causes sickness some other preparation may be substituted for it. It is essential that mercurials should not be employed systematically, for comparatively small quantities are apt to set up salivation in persons who have Bright's disease.

When dropsy cannot be otherwise got rid of, it becomes necessary to resort to *acupuncture*. This is generally done with a needle, which is pushed into the subcutaneous tissue of the thigh, or leg, or foot, and moved about before being withdrawn, so as to make an open channel, through which the fluid may find its way and escape. The surface should be first well greased, in order that maceration of its cuticle may be as much as possible prevented. It is surprising how freely the fluid will often trickle away. It should be soaked up by blankets wrapped round the limb; and these must be frequently changed, being well washed before being used a second time. If four or six punctures are made there is often a great diminution of the anasarca within a day or two; and the abdomen too may appear to be much less swollen than before. Some physicians prefer to make small incisions, or to employ Southey's tubes. The great objection to puncturing the skin is the danger that an erysipeloid inflammation may be set up as a consequence.

For the more acute forms of uræmic seizure, *venæsection* is often the best remedy; the abstraction of ten or even twenty ounces of blood may be followed by a strikingly rapid subsidence of the symptoms. In other cases the inhalation of chloroform proves effectual. It is generally advisable to give a drastic purge; and for this purpose calomel may be used, or elaterium, a drug which, on account of its irritating properties, is better avoided in other circumstances. Sometimes cold affusion to the head may be employed with advantage. If further experience should confirm the idea that the occurrence of uræmia often leads to a greater activity on the part of the kidneys, the conclusion would seem to be that it should not be treated by diaphoretics, such as pilocarpin.

When an acute attack of Bright's disease is passing off, the greatest care must be taken to prevent the occurrence of a relapse. The patient should be kept warmly clad, the use of flannel next the skin being especially insisted on. He must avoid exposure to cold, as well as bodily fatigue and exertion. The diet should be carefully regulated.

At this stage, and also when the disease becomes chronic and is attended with marked anæmia, *ferruginous compounds* are very useful. The tinctura ferri perchloridi is most usually prescribed, but sometimes it is not well borne, so that some milder preparation must be substituted, such as the ferri ammoniæ citratis, or the tinctura ferri acetatis. Digitalis may be given as well; and if there is headache, the bromide of potassium is very useful. It is often of great advantage to the patient to spend the winter in a hot dry climate, such as that of the Riviera, or of Egypt. The diet must still be carefully regulated, as regards both solid food and alcoholic drinks.

Chronic cases.—In renal cirrhosis, the first indication is to control the arterial tension. At first sight the physiological explanation given at p. 450, according to which this is part of a system of conpensation for the kidney disease, might seem to involve the conclusion that it should not be interfered with by treatment. But even from that point of view it must be desirable to relieve the circulating fluid, as far as possible, of any substances which the kidneys may find difficulty in removing, and the accumulation of which renders the excessive pressure necessary. As a matter of fact, the regular administration of *purgatives* is believed by Dr Broadbent and by Dr Mahomed to be of great value, and even to be capable of warding off for a time the occurrence of cerebral hæmorrhage. *Nitroglycerine*, again, is sometimes serviceable; especially, I believe, in relieving uræmic dyspnœa. Or recourse may be had to inhalations of nitrite of amyl, the effect of which is, however, too temporary to be of much assistance.

In cases of cardiac dropsy secondary to renal cirrhosis, *digitalis* is often invaluable. By it the disease can often be kept at bay for a considerable time, and the patient may even be restored to a state of apparent health.

[By such treatment with drugs, combined with strict diet and, if possible, removal to a warm climate during winter and spring, cases of Bright's disease, both parenchymatous and interstitial, may be greatly alleviated and not infrequently cured. The statement made twenty years ago that parenchymatous nephritis when once established is as hopeless as tuberculous phthisis, is certainly untrue now, even with the better prognosis of phthisis itself, which pathology and clinical experience alike have taught us. Dr Wilks has told me how frequently he sees cases recover which formerly he would have condemned.

Even in what is justly regarded as one of the most fatal, as it is one of the rarest forms of Bright's disease, renal cirrhosis in a young subject, I have seen a young man, in whom anæmia, casts, œdema, vascular tension, and (probably) hypertrophy of the left ventricle were present, without gout or other obvious cause, completely recover after a winter spent in Egypt, with the help of flannel, diet, and chalybeate treatment.—Ed.]

TUBERCLE, CANCER, AND OTHER STRUCTURAL DISEASES OF THE KIDNEYS

NEPHRO-PHTHISIS.—*Tubercular nature—Symptoms, causes, and treatment.* MALIGNANT DISEASE.—*Sarcoma—Carcinoma—Symptoms—Nephrectomy.* HYDATIDS OF THE KIDNEY.—*Chyluria and Filaria sanguinis—Endemic hæmaturia and bilharzia—Renal malformations—Floating kidney.*

In the present chapter I have to deal with the more grave structural affections of the kidneys: tuberculous disease, cancer, and other malignant new growths, parasitic affections, and displacements.

TUBERCULOUS DISEASE OF THE KIDNEY. NEPHRO-PHTHISIS.—In cases of acute tuberculosis miliary tubercles are found after death in the kidneys, as in other organs, but there are generally very few of them, at least so far as the naked eye can judge of their numbers. And even when they are numerous they are not known to produce any symptoms, or to affect the course of the disease. Ebstein, however, says that the epithelial cells of the kidneys are generally in a state of advanced granular degeneration, a point of interest in connection with the observations noticed (vol. i, pp. 68 and 79) as to the occurrence of catarrhal changes in the lungs and in the testicles in acute tuberculosis.

The affection now to be described is of a different nature. It consists in the gradual destruction of the kidney, generally of one side only, by the formation of cavities with cheesy walls, which may fairly be called *vomicæ*, from their resemblance to vomicæ in the lungs. And precisely the same differences of opinion have of late years prevailed about this "nephro-phthisis" as about the corresponding pulmonary disease. Some observers have refused to recognise in it any tuberculous character, regarding it as a mere "cheesy inflammation," and declaring that if tubercles are found they are secondary and accidental formations. But I have always felt convinced, from the close analogy which exists between the renal affection and other like affections, occurring not only in the lungs but in many other organs, that they all possess specific characters. And now that, since the discovery of the tubercle bacillus, the truth of this view has been established so far as concerns the so-called "caseous" and "catarrhal" forms of pulmonary phthisis, we may, I think, confidently anticipate that it will before long be universally admitted that the corresponding morbid conditions of the kidney are also essentially tuberculous. The tubercle bacillus has, indeed, been already found in the urine in some cases, and all that seems to be needed is the systematic investigation, not only of the renal secretion, but also of the tissues of the kidney after death, so as to determine whether it is not constantly present.

There have been some differences of opinion as to whether tuberculous disease of the kidney has its starting-point in the cortex or in the medulla of a pyramid. My own observations have convinced me that it may begin in either situation. Perhaps it may be worth while for me to cite briefly the cases bearing upon this question that I have met with, and the more so since they show that, as a rule, tubercles are present from the first, and that even when this is not obvious the character of the morbid process is yet unmistakable.

In 1874 a woman, aged twenty-six, died in Guy's Hospital of phthisis; one kidney had in its cortex a cluster of yellow tubercles, from which a linear yellow streak extended down to the corresponding pyramid. In 1873 a girl, aged six, died of tubercular peritonitis; in one kidney, near the apex of a pyramid, was a round tubercle just beginning to soften; the mucous membrane of the pelvis of the other kidney was covered with tubercles, as was also the lining membrane of the bladder. In the same year another girl, aged four and a half years, died of acute tuberculosis, the bronchial glands being caseous; in a single pyramid of one kidney, not quite reaching either its free surface or its base, there was a well-marked vomica, with an indurated cheesy border. In the same year a man, aged forty, died of phthisis, with caries of the spine; in one kidney there was early tuberculous disease, ulcerating so as to form a conical cavity; in the adjacent part of the cortex there were cheesy grains and nodules up to the size of swan-shot, some extending to the surface of the organ. In 1863 a boy, aged fifteen, who had been admitted for bladder symptoms, died of tubercular meningitis; both kidneys, but especially the right one, contained "softening tuberculous matter, as well as distinct tubercles, regularly arranged in the cortex;" in the mucous membrane of the pelvis of the right kidney there were well-marked isolated tubercles, and also in that of the corresponding ureter and of the bladder near its neck. In the same year a man, aged twenty-two, died of acute general tuberculosis and tubercular meningitis; the kidneys were stuffed with soft yellow tubercles, in some places collected into groups, and apparently about to soften into abscesses; the pelvis of the right kidney was lined with a layer of granular lymph, and this extended down the ureter into the bladder, which itself was affected with tuberculous ulceration; all round the opening of the right ureter into the bladder its mucous membrane was covered with isolated tubercles of various sizes. In 1876 a man, aged thirty-four, died of bronchitis and emphysema, the lungs being quite free from tuberculous lesions; in one kidney a single pyramid was eaten away at the tip, and the rest of it was changed into a gelatinous material of sulphur-yellow colour. In 1875 a man, aged twenty-three, died of pleurisy and of tubercular disease of the lung; in the substance of a single pyramid of one kidney there was an early patch of caseous infiltration. In 1878 a woman, aged twenty-eight, died of phthisis; one kidney contained a circumscribed cheesy mass of the size of a damson, and two of its pyramids were also affected with early tuberculous lesions. In 1879 a youth, aged nineteen, died of spinal disease with psoas abscess; one kidney showed several early tubercular masses excavating the cortex and forming vomicæ with cheesy walls; on the mucous membrane of the pelvis there were also scattered grey tubercles and caseating patches. In 1880 a woman, aged twenty-five, died of bronchitis; in one kidney there were two typical vomicæ with cheesy walls. In the same year a man died of lardaceous disease of the viscera, the result of caries of the spine; in one kidney there were two vomicæ, one in the cortex, the other in a pyramid, with opaque caseating tubercles round them. Another man, also in 1880, died of phthisis; in one kidney a pyramid was eroded by a single tuberculous ulcer, and beyond this, in the cortex, there were opaque white tubercles.

These cases, while they show that the characters of tuberculous disease of the kidney at its commencement vary within certain limits, yet appear to me to prove that the morbid process is always fundamentally the same. They also indicate clearly how close is the relationship between this affection and the tuberculous diseases of other organs, though it is to be observed that in two instances the cause of death was merely bronchitis. When the renal mischief has time to run its course, and to prove directly fatal, it is obviously far less likely to have been preceded by tuberculous lesions elsewhere; but even in such cases they often develop themselves sooner or later, not only in other parts of the urogenital apparatus, but also in the most remote organs.

In a case which I inspected in 1874, an ulcer which excavated a single pyramid of one kidney had a hard calcareous wall, which seemed to indicate that the affection was arrested in its progress, and might have remained stationary had the patient lived. Otherwise, all my observations have tended to confirm the usual opinion that when once tuberculous disease has begun it goes on and destroys the whole substance of the organ. The vomicæ, which correspond more or less accurately with the affected pyramids and the portions of cortex belonging to them, keep increasing in size, their cheesy walls spreading further and further into the renal tissue, until they lie close beneath the capsule and touch one another on all sides, or communicate by lateral openings. At the same time the capsule undergoes great thickening and may become almost as hard as fibro-cartilage. The mucous membrane of the pelvis, from an early period, is converted into a thick whitish-yellow layer. Any parts of the cortex that escape removal by ulceration are converted into a tough, white, fibrous material that shows no trace of a tubular structure, as was well illustrated by a specimen which was shown by Mr Lucas to the Pathological Society, and upon which I reported for the Morbid Growths Committee in 1875. Almost always there is enough of this material to provide septa by which the excavated organ is permanently divided into a series of sacculi which may be more or less completely shut off from one another; and these may at length lose their cheesy walls and become bounded by a smooth lining membrane. Their contents vary in consistency in different cases; sometimes they are full of a cheesy semifluid pulp, sometimes of a substance exactly like putty or mortar, and highly impregnated with lime salts; occasionally some of them ultimately come to contain a transparent yellowish fluid, in which there may be seen floating crystals of cholesterine. It is obvious that such matters could hardly accumulate in large quantity were there a way freely open for their escape. But the fact is that from an early period in the course of the disease the ureter is commonly blocked and impervious. Its mucous membrane undergoes the same change as that which affects the renal pelvis; its other coats are indurated, and it becomes converted into a hard cord, which may be as thick as a pencil, or even as one's finger, and which has little or no hollow space left in its centre. The bladder, in its turn, shares in the morbid process. Sometimes an excavated ulcer first forms round the orifice of the ureter. Sometimes the whole of the vesical cavity becomes lined with patches of tough cheesy deposit, and more or less extensively ulcerated. In some cases, however, the bladder affection begins at the same time with, or even before, that of the kidney. Not infrequently the morbid process extends into the urethra. I have notes of three cases, in each of which it reached as far as the external meatus, where the rough greyish-yellow appearance of the mucous membrane might easily have been seen during the patient's life had attention been directed to it. In one of these cases the canal was extraordinarily widened, so much so that a No. 16 catheter was required to fill it. When the affection is advancing along the urethra at the time of death the part most recently affected sometimes shows very obvious grey tubercles, apparently situated upon the surface of the mucous membrane. Again, in many cases the prostate, the vesiculæ seminales, the vasa deferentia, and the testicles, some or all, take part in the disease. For example, apart altogether from cases in which the patient complains of anything that the surgeon would ordinarily recognise as "strumous disease of the testicle," one or more hard nodules can often be felt in the epididymis if careful search is made for them. As a rule, the vas deferens retains its natural size when affected with tuberculous mischief, but I have notes of one case in which during life the whole spermatic cord appeared obviously indurated. In the prostate the general result of tuberculous disease is to cause moderate enlargement, with the formation of vomicæ having caseous walls; and the same may be said also of the vesiculæ seminales, except that the cavities which are seen in them are formed out of those that are naturally present. Writers say that in female patients it is not usual for the internal genitalia to become affected with tuberculous lesions as a complication of a like affection of the kidneys. But I have notes of only two cases in which such lesions were found in the Fallopian tubes.

Altogether, among some thirty-four cases of tuberculous disease of the kidney which I have found recorded in the reports of post-mortem examinations at Guy's Hospital, and in which the renal affection was so far advanced as to have played a more or less important part in bringing on death, I hardly find a single instance in which the other parts of the uro-genital apparatus beside the kidney itself were entirely free from tubercular lesions. There are very few cases in which the only part diseased besides the kidney was the ureter. It is therefore not a little curious that of the kidneys themselves one alone is very often affected, the other showing no trace of any tuberculous lesions. Among my thirty-four cases this occurred in twenty-two, while in the other twelve the disease was bilateral, but always much older and more advanced on one side than the other. It has been stated that the right kidney is much less liable to become tuberculous than the left; but I find that the relative frequency of the disease on the two sides is only as three to four.

The *symptoms* of tuberculous disease of the kidney are remarkably few. There may be pain in the loin, and it may occasionally be paroxysmal in character. In some cases there is tenderness on pressure. The diseased organ very rarely forms a tumour that can be detected by manipulation of the abdomen. Ebstein alludes to cases in which there has been a mass as large as a child's head. I find no such instance among those of which I have notes. In Mr Lucas's case, already referred to, which occurred in a little girl of seven, a circumscribed tumour was detected in the right hypochondriac and lumbar regions; after death the kidney was found to be six inches in length, and eleven in circumference. In one case at Guy's Hospital a tumour is said to have been felt, but at the post-mortem examination the kidney only weighed eighteen ounces. In another case there was a swelling which for a time led to the suspicion that the disease was malignant; but this ultimately proved to be an abscess behind the kidney, and therefore not due to any considerable enlargement of the organ itself. As a rule, indeed, a tuberculous kidney is but little above the natural size.

The *urine* is sometimes normal, the reason probably being that the ureter is blocked, as already described. It is possible that tuberculous disease may sometimes go on to complete destruction of a kidney without any discharge from it reaching the bladder, so as to mix with the secretion from the opposite kidney. But in most cases the urine is seldom free from pus, or blood. Hæmaturia is not at all constant; among eighteen fatal cases, in which I have notes of the symptoms that had been present during life, I find only ten in which blood is said to have been at any time observed in the urine; and in one or two of these cases there were phosphatic calculi also in the kidneys or in the bladder, and in most of them the bladder was likewise affected with tuberculous disease, so that the exact source of the hæmorrhage was after all doubtful. The most striking case is one of a man who a year before his death was stated to have one day passed a pint of blood *per urethram,* after straining his back in lifting a heavy weight. Pyuria is a far more conspicuous and important symptom. The quantity of pus in the urine is often considerable, forming a thick deposit. Under the microscope a large proportion of the pus-cells are often found to be undergoing disintegration. The sediment may also contain granular amorphous masses of *débris,* insoluble in acetic acid, and even (it is said) elastic fibres and shreds of connective tissue, the presence of which is very significant as regards the nature of the disease. To the recent discovery of the bacillus of tubercle in the urine I have already alluded. In most cases the urine has an acid reaction throughout the whole course of the patient's illness, but sometimes, especially when the bladder is severely affected, it becomes ammoniacal and fœtid. There is then severe dysuria. Other cases differ widely among themselves as regards the presence or absence of vesical symptoms; sometimes pain in micturition and strangury have been conspicuous features of a case during life, but on post-mortem examination the bladder has been found apparently healthy.

Pyrexia is generally present if there are other marked symptoms, and it may assume a hectic type. It is attended with loss of appetite and often with nausea and diarrhœa, and emaciation may set in and bring the disease to a fatal termination.

The duration of such cases from the time when the patient is first discovered to be ill is commonly from six months to two or three years. But it would be a great mistake to suppose that this fairly represents the course of the affection. On the contrary, there are many instances in which its progress is so slow that the opposite kidney has time to become hypertrophied, and probably carries on its function with perfect efficiency. Ultimately this, the healthy kidney, may in its turn suffer from the effects of pressure upon its pyramids, as the result of tuberculous disease of the bladder; and an ascending suppurative nephritis may set in and rapidly bring the case to an end. Or the hypertrophied organ may become affected with Bright's disease, as described at p. 390. Or the drain of pus from the tuberculous kidney or from other parts of the uro-genital apparatus may lead to the development of lardaceous changes, both in the opposite kidney and in the viscera generally. In such circumstances the urine may be albuminous and may contain tube-casts, even though, in consequence of the tuberculous ureter having become obstructed, no pus is for a time being discharged. In one case at Guy's Hospital the immediate cause of death was the extension of ulceration from the tuberculous kidney into the peritoneal cavity. A few cases have ended fatally as the result of the formation of perinephric abscesses, which have pointed in the loin, or have burrowed down in the sheath of the psoas muscle, until they made their way into the hip-joint. Another way in which the disease frequently terminates is by the supervention of phthisis, or tubercular peritonitis or meningitis, or general miliary tuberculosis.

With regard to the *causes* of tubercular disease of the kidney, apart from those that are concerned in the production of tubercular affections in general, very little can be said. It is much more common in men than in women, the proportion being in my cases as three to one. As regards the influence of age, I find, taking only those cases in which the renal disease played a principal part in bringing about the fatal issue, that among twenty-nine cases there were three in which death occurred between the ages of ten and twenty, twelve between twenty-one and thirty, eight between thirty-one and forty, five between forty-one and fifty, and one between fifty-one and sixty.

Dr Roberts has given somewhat different figures, the proportion of cases during the later periods of life being much larger.

In the *treatment* of the disease there is probably not much to be done beyond placing the patient under favourable conditions and giving cod-liver oil and tonics. At an early stage, if the diagnosis could be made with certainty before the lower part of the ureter and the bladder had become affected, it might probably be advisable to run the risk of performing nephrectomy. Mr Morrant Baker ('Trans. Internat. Congr. 1881,' vol. ii, p. 262) performed this operation in the case of a girl aged seven, with a result so far successful that five months afterwards the child had greatly improved in health, being able to play all day long, and going out of doors frequently for a walk. Her illness had begun with hæmaturia about twenty-two months before the kidney was excised. There was alarming faintness at the time of the operation, especially when the pedicle was ligatured. In this case pus continued to be discharged with the urine at the date of the last report, so that it was feared that disease still existed in the ureter or even in the bladder. In vol. xv of the 'Clinical Society's Transactions,' Dr Goodhart and Mr Golding Bird have recorded a case in which the right kidney was extirpated for tuberculous disease, and which proved fatal about four hours after the completion of the operation. The patient had been seriously ill for about ten weeks, but had complained of pain in the back for eighteen months. At the autopsy the ureter was found to be diseased in its whole length, there being ulceration in its vesical orifice, and the mucous membrane of the bladder was thickened and opaque. In the prostate there was some cretaceous (? strumous) material. It is the liability of the disease to affect other parts of the urogenital apparatus that seems to me to render nephrectomy a doubtful expedient in such cases.

SARCOMA AND CARCINOMA OF THE KIDNEYS.—When a malignant growth, in whatever part of the body it may be situated, gives rise to abundant secondary nodules in distant organs by infection from the blood-current it not infrequently happens that some of them are seated in the kidneys. But in such cases the renal affection is seldom of clinical significance, except sometimes by causing more or less hæmaturia, or (if the primary growth is melanotic) by giving to the urine a brown or black colour.

Primary malignant tumours of the kidney are decidedly rare. Statistics from various sources are cited by Ebstein (in Ziemssen's 'Handbuch') in proof of this fact, and they are fully borne out by the reports of post-mortem examinations at Guy's Hospital, where I find only about fifteen cases during a period of twenty-two years.

Until recently, two distinct affections were included under the name of "malignant disease," or "cancer" of the kidney. One of them occurs chiefly in infants and young children up to the age of eight or ten years, though I have notes of two cases in boys aged respectively eleven and seventeen, and I do not doubt that it is sometimes seen in early adult life. This, although Ebstein speaks of it as a carcinoma, is, I believe, always really a sarcoma, like most of the malignant growths of other parts that are met with in very young subjects. It forms a smooth rounded mass, which sometimes reaches an enormous size, weighing ten, twenty, or even thirty pounds, so that I remember to have read of one autopsy at which it was said that instead of the tumour being removed from the child's body, the body was removed from the tumour. Such a growth is commonly very soft and elastic, and may even appear to fluctuate, and it is therefore very likely to undergo puncture at the hands of the surgeon, a procedure which is generally harmless, but which I once saw followed by a sharp attack of peritonitis. Sometimes, besides blood, a small piece of sarcomatous tissue is brought away in the orifice of the trocar. These sarcomata of the kidney are very vascular, and hæmorrhage often takes place into their substance, causing a sudden increase in their size. They grow, too, with great rapidity, destroying life sometimes within a few weeks, and almost always in less than a year, from the time when they are first discovered. They affect one kidney only, but Dr Abercrombie recently showed to the Pathological Society three cases, occurring in young children, in each of which both kidneys were invaded at the hilus by a sarcomatous growth, which, however, did not produce a tumour that could be recognised clinically. A further point to be noted is that a sarcomatous tumour occurring in a young child in the position of the kidney is not always really seated in that organ. Some years ago Dr Dickinson brought before the Pathological Society (vol. xxi, p. 397) a specimen in which the growth occupied the lumbar glands and merely pushed the kidney before it, and in a case of Dr Day's it affected only the capsule of the kidney, this itself being healthy.

The other form of primary malignant disease of the kidney is a true *carcinoma.* It is seen chiefly in persons past middle age; in almost every instance that has occurred at Guy's Hospital within the last twenty years the patient has been more than forty-five years old. It is much more common in men than in women. It does not generally grow very large, perhaps not exceeding the size of a cocoa-nut; but it may sometimes occupy all one side of the abdomen, or even appear to fill the whole cavity, like a great ovarian tumour in a woman.

In such cases, however, a good part of its bulk is commonly made up of hollow spaces containing a blood-stained fluid; or there may be a large accumulation of a similar fluid in the dilated renal pelvis, this being shut off from the ureter. Carcinomata of the kidney are often spoken of as scirrhous, but I think that they seldom deserve that name, being of not more than medium hardness. In very rare instances they have been found to have undergone colloid degeneration. In 1876, I showed to the Pathological Society (vol. xxvii, p. 204) a "carcinoma lipomatosum" of the kidney—a growth which looked like adipose tissue, but which had extensively invaded the renal veins as well as the substance of the organ, and which had a typical alveolar structure, the alveoli being filled with large cells loaded with fat. The specimen had no clinical interest, for the patient died of another disease. Sometimes carcinoma of the kidney causes a a general enlargement of the whole organ, the distinction between cortex and pyramids being still traceable in the tumour; sometimes only certain parts are affected, there being rounded or irregular masses of growth, with intervening tracts of healthy tissue. Following Waldeyer, most pathologists now believe that the renal epithelium is the starting-point of the growth.

With regard to the *causes* of cancer of the kidney very little is known. In a few cases it has appeared to be the result of a blow or of a kick in the loin; but one may doubt whether it was not really present before, and whether the injury did more than bring on symptoms, such as hæmaturia, by which attention was first drawn to it. I have, however, notes of two cases, in each of which the disease appeared to be superimposed upon another affection of older date. One occurred in a man of forty-five, who was said to have been troubled with his water for twenty or thirty years, passing a gelatinous substance with it. The right kidney was found to have its calyces dilated into a number of chambers, and in the pelvis lay a large irregularly branched calculus, "like a knotted branch of a tree." Growing from the upper part of the organ was a cancerous mass, which also extended upwards behind the liver and penetrated through the diaphragm into the lung. The other case was that of a man, aged sixty-six, who had had hæmaturia twenty years previously, and who came under treatment for a recurrence of this symptom ten months before his death. The lower part of the kidney showed the ordinary appearances of "hydronephrosis," or "sacculation" from distension of the calyces; but into many of the sacculi soft masses of a carcinomatous growth were projecting; and the upper part of the organ formed a solid tumour. Even apart from the post-mortem appearances, it is, I think, almost inconceivable that the cancer should in this patient have dated back as far as the earliest attack of hæmaturia, although Ebstein speaks of a case of renal cancer which was believed to have lasted for eighteen years. The occurrence of a calculus in the kidney as a complication of malignant growths is mentioned by writers as not very frequent, and sometimes, perhaps, a phosphatic stone may be of later development than the tumour, especially if pyelitis happens to be present. The ordinary duration of cases of carcinoma of the kidney in adults is probably from six months to two years after the first appearance of symptoms.

The *symptoms* of a malignant growth in the kidney, whether sarcomatous or carcinomatous, are mainly three: the presence of an abdominal tumour, hæmaturia, and pain.

1. The *tumour* has the usual characters of tumours seated in the kidney. It occupies one side of the abdomen, having its centre opposite the lumbar region, between the lower ribs and the iliac crest. It often bulges into the loin, and one can move it slightly forwards by pressing the loin with one hand, while the other is placed over the front of the abdomen. It does not descend during inspiration, and the fingers can be inserted between it and the rib cartilages, showing that it is not seated in either the liver (if on the right side) or the spleen (if on the left). It is sometimes perfectly smooth and uniform, sometimes more or less uneven and lobulated. Overlying it in front there is commonly a part of the colon, the hepatic or the splenic flexure, according to the side affected, which either may be felt as a ridge, bending nearly at a right angle, or may be traced by its tympanitic percussion-sound contrasting with the dull sound obtained elsewhere. Sir Spencer Wells has proposed, in doubtful cases, to inflate the rectum with air, so as to render the position of the bowel more conspicuous. In one case that I saw some years ago, besides the colon, I could feel several coils of small intestine in front of the growth; they were freely moveable, and could be made to slip away from beneath the finger under manipulation. Dr Roberts relates a remarkable instance in which not only was the stomach made out during life to lie in front of a cancerous left kidney, but the spleen could be distinctly felt as a separate mass in the iliac fossa, lying over the lower and inner part of the tumour. Mr Holmes recorded in vol. xxiv of the 'Pathological Transactions' a case in which a malignant growth of the kidney pulsated and was attended with a bruit, so that aneurysm was suspected. There often is considerable distension of the superficial abdominal veins. This probably may be due to compression of the inferior vena cava by the tumour, or by enlarged glands. But in many instances the growth fungates into the renal vein, and it may even protrude into the cava so as to narrow its calibre. A further result of such conditions is that the feet and legs become œdematous; and Rindfleisch speaks of embolism of the pulmonary artery as being sometimes caused by the detachment of portions of the cancerous thrombus. In a case that was observed at Guy's Hospital in 1871 the disease made its way into one of the veins of the colon, and thence into the portal vein and its branches within the liver; the result was the occurrence of ascites in large amount.

2. *Hæmaturia* is by no means a constant symptom. Ebstein found it absent in twenty-eight out of fifty-two cases collected by him. Very often it is the earliest indication that anything is amiss with the patient. Some-

times it is directly brought on by a blow or fall. It may recur again and again at irregular intervals for a considerable time before any tumour can be detected. It is then apt to be set down to a renal calculus, but one distinction is that the hæmaturia is not generally attended with a marked increase or aggravation of pain. In many cases the bleeding comes from portions of the growth that protrude into the pelvis of the kidney. But in other instances its source is from a tumour within the cortex, and if such is the case tube-casts containing blood-corpuscles may doubtless be found in the urine, as is stated by Ebstein.

3. The *pain* produced by a malignant tumour of the kidney is very variable in degree; sometimes it is altogether absent. Its usual seat is in one lumbar or hypochondriac region, but it may radiate widely over the lower part of the chest to the front of the abdomen, or to the crista ilii, and even down the thigh. It may be either a constant, dull aching, or paroxysmal, sharp, and cutting in character. Sometimes there is much tenderness to pressure. The pain is seldom, if ever, attended with retraction of the testicle, in which respect it differs from the pain due to calculus. If, however, clots of blood formed in the pelvis of the kidney should become impacted in the ureter, the pain may assume a different character and become exactly like that which accompanies an attack of renal colic.

Other symptoms that may be present in cases of malignant growth in the kidney are anorexia, nausea, vomiting, and constipation or diarrhœa. In children, however, it is said that there is sometimes a voracious appetite with great thirst. The patient usually becomes rapidly wasted, anæmic, and cachectic. The temperature remains normal or subnormal, and the pulse may be unduly slow. Death is usually attributable to exhaustion, and is sometimes preceded for a few days by stupor or insensibility. In a case recorded by Bright in the first volume of the 'Guy's Hospital Reports,' 1836, the tumour gave way into the abdominal cavity, causing a large extravasation of blood. Some years ago a woman was admitted into hospital for paraplegia, which had been coming on during two months, but she was said to have had hæmaturia four months before and to have been ill for a year; at the autopsy it was found that there was a primary cancer of the left kidney, and that the growth had extended into the spinal canal. A like case has been observed by Cornil. In a patient who died in Guy's hospital in 1870 all the symptoms were cerebral, and the immediate cause of death was the presence of secondary tumours in the brain.

It can easily be understood from the foregoing description that the *diagnosis* of malignant disease of the kidney is often difficult, or even impossible. One of the most striking instances that I remember is that of a man, aged thirty, who died in Guy's Hospital many years ago of wasting and weakness and anæmia, and whose case excited great interest from there being no discoverable local symptoms. At the autopsy it was found that the right kidney was the seat of a primary growth which had destroyed nearly its whole substance, but which did not reach its pelvis.

Treatment.—It is obviously improbable that any very large measure of success will ever be attained by nephrectomy in cases of sarcoma or carcinoma of the kidney. But some few cases have already turned out much more favourably than could have been anticipated. In 1877 Mr Jessop, of Leeds, removed an encephaloid tumour of the kidney from a boy two and a half years old; rapid recovery took place, but about eight months afterwards the disease returned, probably in the lumbar glands, and the case ended fatally a few weeks later. In 1878, Martin, of Berlin, extirpated a sarcomaof the kidney, weighing twenty-eight ounces, in the case of a woman, aged fifty-three; she was up on the eighth day, and went home on the seventeenth day; Czerny speaks of her as being still well two years afterwards. In 1879 Lossen, of Heidelberg, performed nephrectomy in a woman, aged thirty-seven, for an "angio-sarcoma" of the right kidney which, being moveable, was mistaken for an ovarian tumour; she recovered in six weeks, and she continued to be in good health eighteen months later. In 1881 Czerny removed a large vascular sarcoma from a man, aged fifty-three, who at the time of the operation was very cachectic, and suffered greatly from vomiting; two months later he left the hospital in a state of blooming health, with the proportion of red discs in his blood twice as great as it had been previously. Against these successes, however, must be set down a good many cases in which the extirpation of malignant growths in the kidneys has either been attempted ineffectually, or has proved rapidly fatal by shock or by peritonitis. (See Czerny's tabular statement in the 'Trans. Internat. Congress, 1881,' vol. ii, p. 249.)

Apart from surgical interference, the only treatment is to maintain the patient's strength by tonics, and to relieve the symptoms from which he may suffer.

Hydatid of the Kidney.—According to Davaine the kidney comes third in the order of frequency among the organs liable to be infested with the echinococcus in its encysted stage. In the records of post-mortem examinations at Guy's Hospital I find, however, only three instances of such an affection: in one the parasite was of the size of a plum; in another of an orange; in the third it formed a bulging elastic swelling extending from the left hypochondrium into the loin, and containing two pints of fluid. Clinically, the diagnosis must generally be based upon the recognition of an abdominal tumour, having the characters that belong to tumours of the kidney, more or less tense and rounded in form, and possibly yielding the characteristic *fremitus* to percussion. The diseases for which it is most likely to be mistaken are simple renal cyst, hydronephrosis, soft sarcoma of the kidney, and (in the female) cystic disease of the ovary, as in a case in which Spiegelberg performed an operation which he intended for ovariotomy. A point which may sometimes aid in the diagnosis is the discovery of a second hydatid in the liver; this was the case, for example, in the patient whom I mentioned (p. 296) as having died at Guy's Hospital with a large hydatid in the kidney holding two pints of fluid. In many instances a hydatid in the kidney probably remains for years—perhaps from childhood or middle age throughout the entire life of the individual—without affecting the health. Or it may die, and dry up into a pultaceous or cheesy mass, which henceforth possesses no power of doing any damage. But in the majority of cases (if one may judge from published records) it sooner or later ruptures into the pelvis of the kidney, after which the daughter-cysts and scolices pass down the ureter and are expelled with the urine. I have already referred to such cases as presenting the symptoms of renal colic (see p. 386). It must be borne in mind that the presence of cysts or hooklets in the urine is not in itself proof of the existence of a hydatid in the kidney. Nearly, if not quite as frequent a seat of the parasite is the pelvic pouch of the peritoneum, and in such cases it sometimes ulcerates directly into the posterior wall of the bladder. I have seen two examples of pelvic hydatids, and in each instance there was a tumour in the hypogastric region of the abdomen, having exactly the shape and the other characters of a distended bladder. In cases of renal hydatid, the passage of daughter-cysts down the ureter is often the first indication that anything is amiss with the patient; and when the parent cyst is small, no tumour may be discoverable in the loin. Not infrequently the rupture of the parasite is directly produced by a blow or by a fall; or the symptoms may appear to be brought on by riding or by driving, as in cases of renal calculus. Sometimes there is only a single discharge of daughter-cysts in the urine, the patient afterwards remaining perfectly well. Sometimes the same thing recurs again and again, at intervals of months or years, during a period of ten, twenty, or even thirty years. Suppuration within the capsule of the cyst sometimes occurs, and blood and pus may be voided in the urine. But Dr Roberts, however, says that the ultimate prognosis is generally favourable. Of sixty-three cases which he collected only nineteen were known to have ended fatally; and in nine of these the cause of death was some disease not directly connected with the renal affection. In some instances, there has been ulceration through the diaphragm, with escape of daughter-cysts into a bronchial tube and expectoration through the air-passages; the prognosis is then very unfavourable. The proper treatment of hydatid of the kidney, when a tumour can be detected, is puncture with a very fine tubular needle, fitted to an aspirator. In two cases Dr Roberts found that the withdrawal of only a drachm or two of fluid sufficed to destroy the life of the parasite, and caused it to pass very gradually into obsolescence and absorption.

CHYLURIA.—A very remarkable condition of the urine, first described by Prout, is one in which it looks white and milky when passed, and soon afterwards sets more or less completely into a soft jelly, like *blanc-mange*, which may take the shape of the vessel that contains it. Sometimes it solidifies within the bladder, and the result may be that there is pain and difficulty in micturition from obstruction of the urethra. The coagulum after a little while liquefies again, a material, somewhat like cream, collects upon the surface, and there falls to the bottom a deposit which is generally of a pinkish colour, from the presence in it of a small quantity of blood. It was recognised by Prout that the characters of the affection were exactly such as might be due to the admixture of chyle with the renal secretion. And this view was confirmed by microscopical examination, which showed that the cause of the opacity was a finely granular material, not large fat-globules as in milk; and also by the application of chemical tests, for by ether a large quantity of fat was extracted, and the urine was also shown to contain albumen in considerable amount. It was further noticed that the peculiar state of the urine was often far more marked a few hours after a full meal than when the patient had been fasting for some time.

Until very recently, however, the pathology of chyluria has been a complete mystery, and there is scarcely anything more curious in the history of medicine than the way in which it has step by step been elucidated, until we now seem to know nearly all about it. Prout noticed that a large number of those affected were born, or had lived for many years, in hot climates. Next, in 1866 Wucherer, in Brazil, detected in chylous urine certain minute living organisms, evidently the embryos of a nematode worm. Six years later, in 1872, Dr T. R. Lewis, in India, discovered similar embryos in the blood. It was very soon found that this was no isolated occurrence, and that the hæmatozoon (as it was called) was by no means limited to persons affected with chyluria. In South China, for instance, among 1000 natives taken at random about 100 are said to be infested with this parasite. In that country similar larval entozoa are very commonly seen in the blood of dogs, and in that of many species of birds, so that their presence in man excited the less surprise in the minds of experienced helminthologists. Among those persons who harbour the *Filaria sanguinis hominis* (as Lewis termed it) some appear to be in perfect health, but others are affected with one or more of a limited number of diseases, of which the chief are lymphangitis with varicosity of the lymph-channels in the inguinal glands, lymph-scrotum, elephantiasis of the scrotum or of leg, and chyluria.

Before attempting to show how these various diseases are related to the parasite, which is thus common to them all, I must briefly describe what has been ascertained with regard to its life-history. The first step in the inquiry was the discovery of the parent worm from which the embryos found in the blood are derived. This was effected in 1876 by Dr Bancroft, of Brisbane, in Australia. He first obtained a dead specimen from a lymphatic abscess in the arm, and afterwards four living ones from a hydrocele of the spermatic cord. These he sent to England to Dr Cobbold, who gave to the entozoon the name of *Filaria Bancrofti.* They were all females, and as yet no perfect specimen of a male seems to have been found. The length of the female is from three to three and a half inches; its breath from $\frac{1}{100}''$ to $\frac{1}{90}''$. It has a simple circular mouth, destitute of papillæ, a narrow neck, and a bluntly-pointed tail. Its body is smooth, and of an opaline appearance, and it has been described by Dr Manson, of Amoy, in China, as looking "like a delicate thread of catgut, animated, and wriggling." This observer, in 1880, in operating on a case of lymph-scrotum, removed at the same time a portion of a living worm, and showed that it lay in the interior of a dilated lymphatic. He also proved that the parasite is naturally viviparous, for he saw fully-formed embryos, exactly like those which are found in the blood, escaping from the animal's vagina. Strictly speaking, indeed, the accuracy of this last statement is open to question. For it has been known from the first that the embryo in the blood is always enclosed in a delicate sac or sheath, which fits it accurately, except that a collapsed or unoccupied part is seen projecting like a lash beyond either the head or the tail, according to the direction in which the worm happens to be moving; and it seems now to be certain that this sheath is nothing else than the envelope or shell of the ovum, which, as the embryo develops, yields before it, and so continues to be stretched out over it. Still, the fact remains—and it is one of which we shall presently see the importance—that the parent filaria, instead of throwing off oval passive eggs, gives birth to active organisms, capable from the first of vigorous spontaneous movements. The size of these embryos is such as not at all to interfere with their traversing the lymph-paths through any glands that may come in their way, and so passing on from the lymphatic vessel in which the parent worm lies into the thoracic duct, and beyond this into the blood-vessels. Their diameter, indeed, is only about $\frac{1}{3200}''$, not more than that of the leucocytes which circulate through the lymph-glands; their length is $\frac{1}{90}''$, or rather more.

So long as the embryos of the filaria remain in the blood they continue to be of the same size, and show no indication of undergoing further structural development. This fact is of itself sufficient to suggest to anyone acquainted with helminthology the idea that they are waiting to be transferred to some other host. And it seems to have occurred both to Dr Bancroft and to Dr Manson, independently of one another, that this host might probably be some species of mosquito which feeds on human blood. Dr Manson remarks that the limitation of the parasite to certain parts of the earth's surface was almost sufficient to exclude from his consideration many blood-sucking animals, such as fleas, lice, bugs, and leeches, which are found pretty well everywhere. He therefore came to the conclusion, in 1877, that it was likely to be either the mosquito or the sandfly that took the embryo filaria from its human host, and supplied to it the conditions requisite for its development into a more mature form. Had he been at that time aware of another extraordinary fact that he afterwards discovered he might safely have set aside the sandfly also. This fact is that instead of the young filariæ being found in the blood throughout the whole twenty-four hours, none of them can generally be detected in it during the day, even when they are abundantly present in it during the night. At about 6 or 8 p.m. they begin to make their appearance; by midnight their numbers reach the maximum; as morning approaches they become fewer and fewer; by 8 or 9 a.m. they cease to be discoverable. What becomes of them in the interval is not at present known, but it must be borne in mind that all that has as yet been proved is that the capillary blood-vessels of the integument contain them at night, but not in the daytime. There seems to be no necessity for supposing that they circulate with the blood like its normal constituents, the red discs and leucocytes. Possibly during one part of the twenty-four hours they may be all collected in the pulmonary capillaries, or in those of the deeper structures generally, but when the patient retires to rest they may betake themselves to the vessels of the skin. In a case which Dr Stephen Mackenzie has recorded in the 'Pathological Transactions' for 1882 observations at intervals of three hours were made for weeks together, and the periodicity was found to be as complete as it possibly could be. Dr Mackenzie also submitted his patient to the experiment of having his habits of life reversed, so that for nearly three weeks he remained out of bed all night, and rested in the daytime, the hours of his meals being arranged accordingly; the result was that during this time the filariæ were found in the blood during the day, but not at all, or only in much smaller numbers, during the night. Obviously, therefore, that which determines their migrations is the resting or moving condition of their human host. But it is nevertheless impossible not to recognise the fact that their usual habit of entering the blood (or perhaps, rather the capillaries of the integument) at night-time is precisely adapted to bring them within reach of the proboscis of a nocturnal blood-sucker, like the mosquito. In Dr Mackenzie's case the blood seems always to have been taken from the patient's finger; as much of it was examined as would lie beneath a five-eighths inch cover-glass. In this quantity of blood, at midnight, there were often fifty or sixty, and sometimes even eighty or ninety filariæ. Dr Mackenzie therefore calculated that from thirty-six to forty millions of them were probably present in the whole mass of circulating fluid, but it is obvious that this estimate would have to be enormously reduced, if it should be thought that it is only the capillary vessels of the surface of the body that contain them in such abundance.

Dr Manson lost no time in verifying his hypothesis with regard to the mosquito. He pursuaded a Chinaman, known to be infested with the filaria, to sleep in a "mosquito house." Next morning the gorged insects were caught and examined. The blood in their stomachs was found to contain filariæ in even larger numbers than that of the man from whom it had been derived. It is supposed that they become entangled by their lashes in the proboscis of the mosquito and are so removed from the blood-vessels. Having reached their new host, some of them proceed to enter upon a process of development. They lose their sheaths, grow to the length of one thirtieth of an inch, and acquire a distinct alimentary canal, a mouth crowned with three or four nipple-like papillæ, and rudiments of generative organs. Their movements also become extremely active. These changes are completed in from four to six days. During this time the mosquito, having retired to a shady place, has been digesting her solitary meal, and maturing her own ova; she is now prepared to deposit them upon the surface of water; after which, her career being finished, she dies, and probably falls into the water on which her eggs are laid. The progress of the filaria has not been directly traced further. But there can be little doubt that it makes its way out of the body of the dead insect into the water. And in all probability the next step is that it is swallowed by a human being, from whose stomach it bores a passage into the thoracic duct or into some lymphatic vessel; and along this it then works up stream, in obedience to some strange instinct, until it reaches a spot which it takes for its permanent abode. Here we must suppose that it is joined by another parasite of the opposite sex, after which it proceeds to furnish to the lymph-channels and to the blood-current of its host those swarms of larvæ which formed the starting-point of our inquiry as to the life-history of the entozoon. How long the parent worm lives we do not as yet know; but a case observed by Dr Manson shows that it may be at least as long as thirty-two years; he found living filariæ in the blood of a man aged fifty who had had lymph-scrotum from the age of eighteen. There is some reason for supposing that it may be killed by the occurrence of severe acute disease in the host. At any rate Dr Stephen Mackenzie's patient was attacked with rigors as the result of going out of the hospital on a cold and windy day in October, and after the following day no embryos were ever discoverable in his blood. Pleurisy set in, and an abscess formed near the left collar-bone; and when he died two and a half months later no trace of the worm could be found. Dr Mackenzie supposes that it became dislodged during the rigor, and reaching the termination of the thoracic duct on the left side of the neck, excited both the pectoral abscess and also the pleurisy. However this may be, it seems clearly to have perished from an early period of the man's fatal illness, and its body must be supposed to have undergone disintegration.

It now becomes an important question to determine how it is that the filaria produces such affections as chyluria, lymph-scrotum, and elephantiasis. And, so far as I know, the only hypothesis that can be said to account in any way for such results is one that has been formulated by Dr Manson, in a paper in the 'Pathological Transactions' for 1882. His idea is that so long as the discharge of embryos goes on after the manner above described, the parasite is perfectly innocuous to its host. But from some cause or other it happens in certain cases that, instead of the larval filariæ enclosed in their sheaths, ova in a much earlier stage of development, with unstretched shells, are extruded from the maternal vagina. Dr Manson has twice obtained such ova from the lymphatics; and probably they have been found in the urine also. Now, according to Dr Manson, they measure $\frac{1}{750}''$ in breadth by $\frac{1}{500}''$ in length; according to Dr Cobbold, $\frac{1}{1650}''$ by $\frac{1}{1000}''$. In either case their transverse diameter is far greater than that of the embryos; and nothing is more likely than that they should fail to pass along channels which the embryos would find no difficulty in traversing. Dr Manson supposes, for instance, that when they are carried by the lymph-stream to a gland they become impacted in the small channels formed by the afferent vessel. The necessary result must be, as anastomosing paths become one after another obstructed, a more or less complete stasis of lymph, not only in the neighbourhood of the spot where the parent worm is situated, but also in the whole of one or both of the lower limbs, and also in the scrotum. There seems to be no difficulty in admitting that the ova may be conveyed across from one groin to the other. Dr Manson remarks that this explains why in one-sided elephantiasis the inguinal glands on both sides are often enlarged.

Lymph-scrotum consists in the formation of vesicles which are in fact dilated lymphatics, and which discharge fluid either clear or milky in appearance, according as it is derived from a peripheral vessel, or from one which had already passed through a lymph-gland. The tissues of the scrotum are thickened, but feel soft and spongy. Sometimes similar vesicles form on the inner side of the thigh. In the course of years the flow of lymph commonly ceases; and the scrotum or the lower limb passes into a state of elephantiasis. Or the elephantiasis may be developed in the first instance, there being at no time any vesicles or escape of fluid.

When chyluria occurs, there can be no doubt distended lymph-vessels open upon the surface of some part of the urinary mucous membrane; but whether this usually takes place in the bladder, or in the ureter, or in the renal pelvis, has not yet been determined. One peculiarity of the affection to which I have not yet adverted, is that it is often intermittent, the urine from time to time losing its abnormal characters for days or weeks together. In a case recorded by Ackermann ('Deutsch. Klin.,' 1863) the patient always passed normal urine after he had been lying on his right side for a time. It is to be noted that chyle, in a strict sense of the term, does not exist in the urine in every case in which that fluid is more or less opaline in appearance; if the clot which forms is almost translucent, the cause of it may be merely the presence of lymph that has passed through one or more glands on its way upwards to the thoracic duct. But if the clot is opaque like blanc-mange, and if the state of the urine varies in relation to the patient's meals, there can be no doubt that the obstruction really involves lacteals coming from the intestines. The autopsy upon Dr Stephen Mackenzie's patient showed a large mass of dilated lymph-sinuses and glands, extending from the bifurcation of the aorta below to the diaphragm above, and occupying the whole of the space between the kidneys. The lower part of the thoracic duct was sinuous and much pouched, varying in diameter from three eighths to half an inch. About three inches above the diaphragm it became impervious, and was lost in a quantity of tough, dense material, apparently of inflammatory origin.

These interesting observations as to the relations between the filaria and the chyluria of hot climates of course leave quite undetermined the pathology of the disease when it occurs in persons who have always resided in England or in the north of Europe. Four well-authenticated instances of this are cited by Dr Roberts. It seems not impossible that there may be other causes of obstruction of the lymph-channels of the lower part of the body, which may produce like effects.*

It is obvious from what has been stated with regard to the life-history of the filaria that the *prevention* of the diseases due to this parasite is probably a very simple matter. Dr Manson suggests that wells and water-jars should be covered with a netting sufficiently fine to prevent the entrance of mosquitoes, but it must surely be a better plan to drink no water which has not been boiled or filtered. Care must also be taken to have all raw vegetables thoroughly washed with boiled or filtered water before eating them. In the *treatment* of chyluria, when it is once established, very little can be hoped for from medicines. But perhaps it is worth while to remember that Dr Bence Jones thought that by giving gallic acid to the amount of two drachms daily, he was sometimes successful in restoring the urine to a normal state, at least for periods of several months at a time. When the loss of chyle is considerable, it seems sometimes to cause emaciation and debility, as well as a craving appetite and urgent thirst. In such cases the patient's strength must of course be maintained as far as possible, and he must be impressed with the importance of rest, for exercise is found to aggravate the complaint. The duration of the disease is often very long. Dr Roberts cites two instances, in one of which it continued for twenty-eight years, and in the other for more than fifty years. If death occurs during its course, the cause is generally some intercurrent malady, such as phthisis or Bright's disease.

ENDEMIC HÆMATURIA.—As far back as 1812 Chanotin made known the prevalence of an endemic form of hæmaturia in Mauritius; and subsequent writers afterwards noticed the occurrence of a similar affection in other hot climates. But nothing was made out with regard to its nature, until in

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* [See an admirable case of chylous ascites recorded by Dr Whitla of Belfast, in the 'British Medical Journal,' May 30, 1885.—ED.]

1851 Bilharz, being engaged with Griesinger in investigating the diseases of Egypt, discovered in certain of the veins of the abdominal viscera a trematode worm, to which he assigned the name of *Distoma hæmatobium.* It was found that this parasite gave rise, in some cases, to more or less severe urinary symptoms, and Griesinger, in the 'Arch. d. Heilkunde' for 1854, suggested that it might probably be the cause of the endemic hæmaturia of other countries. Afterwards it was shown to be generically distinct from the flukes, and Dr Cobbold proposed for it the name of *Bilharzia hæmatobia,* which is now generally adopted. In 1863 Dr John Harley detected the ova of the same entozoon in large numbers in the urine of a man who had become affected with hæmaturia at the Cape of Good Hope; and he has since shown (in papers in the 'Med.-Chir. Transactions') that the complaint prevails not only at Nitenhage and at Port Elizabeth in Cape Colony, but also along the coast of Natal.

The Bilharzia is a soft milk-white creature, which differs in shape in the two sexes. The male, half an inch in length, is flattened; but the hinder part of its body acquires a cylindrical appearance from its edges being thinned and folded inwards so as to overlap one another, forming a hollow channel, within which the female lies during congress. The female, three quarters of an inch long, is slender and filiform. The ova are about $\frac{1}{170}''$ in length, and have a sharp projecting beak-like spine, placed usually at one end, but sometimes laterally. According to Dr Zancarol, of Alexandria ('Path. Trans.,' xxxiii), ova with lateral spines are found only when the seat of the parasite is in the veins of the intestine, whereas when it occupies the veins of the urinary tract the spines are terminal; and this statement seems to correspond with observations made by previous writers, though I am not aware that they had definitely recognised such a distinction. (See the drawings by Dr Cavafy, p. 410.) It often happens that empty eggshells are found in the interior of the human body, so that there can be no doubt that the ova may be hatched while in the tissues. But in urine they are said by Dr Cobbold (though Dr Roberts maintains the contrary), never to give exit to the embryos which are often to be seen in them in a fully-developed condition, and quite ready to escape. Such ova, however, when placed in water, become ruptured in a few minutes. The embryos are covered all over with cilia, those around the neck being especially large, and they swim about with great activity.

The further steps in the life-history of the Bilharzia have not yet been ascertained; but the presumption is that the embryo finds in some freshwater mollusc an "intermediate host," and there develops a Cercaria-form. Leuckart seems to think that the most probable way in which human beings become infected is by their swallowing encysted cercaria in the substance of snails or insects eaten accidentally with raw vegetables. Griesinger was of opinion that in Egypt the chief danger perhaps lay in the use of fish as food, though he also mentioned the drinking of Nile water as possibly affording to the creature a mode of entrance into the human body, in which case one must suppose that it is capable of developing directly from the free cercaria up into the sexually mature condition. Dr Harley has been inclined to think that bathing in the rivers of Natal may possibly bring bathers into contact with the parasite, which may either make its way through the skin, or even pass straight into the interior of the bladder through the orifice of the urethra. Males are much more liable to suffer from the Bilharzia than females. The resulting hæmaturia commonly appears during boyhood, but not under five or six years old. It may, however, occur at any age, for one of Dr Harley's patients was a man of seventy-six.

Endemic hæmaturia appears to be mainly vesical in its origin. At the end of micturition, the urine having been quite clear, the patient voids a small quantity—perhaps a teaspoonful or less—of dark blood. Or instead of the blood there may be soft shreds or filaments of mucus, by which the urethra is sometimes blocked for a few minutes; in these shreds the ova of the Bilharzia are found in large numbers. Sometimes a little pain is experienced in the loins or in the perinæum, especially after active exercise, which also increases the amount of blood. The health, as a rule, remains good, though a feeling of lassitude is sometimes complained of, and a more or less marked degree of anæmia may be developed. Boys in Natal appear to take no notice of the complaint, and about the age of puberty it commonly ceases. Long after this, however, Dr Harley has found that ova are still present in the urine, although the patient may imagine that he is quite free from his disorder. And what is very remarkable is that during early adult life he often begins to pass small calculi, in the centre of which the remains of ova may be detected. Indeed, in Egypt the affection commonly leads to the formation of large stones, which have to be removed by surgical operation.

Our knowledge of the lesions produced in the viscera by the Bilharzia appears still to be based almost entirely upon the investigations made by Bilharz and Griesinger in Egypt; they found it present in no fewer than 117 out of 363 autopsies. However, I think it is fair to assume that the state of the bladder in persons who (like the boys of Natal) suffer comparatively little from this parasite, must correspond only with the slightest cases examined by the two German observers. They give a much more serious account of the symptoms caused by it, describing them as like those of a chronic cystitis. The earliest morbid change in the bladder was the formation of swollen, hyperæmic, ecchymosed patches, varying in size up to that of a shilling, and generally coated with tough mucus or with a layer of soft greyish-yellow exudation; they were often limited to the posterior wall of the organ. But in many cases there were also thick deposits of a buttery or soft and granular material upon the surface of the mucous membrane; these were generally encrusted with uric acid and with urinary salts. And sometimes there were raised warty vegetations or fungous-like excrescences, due chiefly to a swollen infiltrated condition of the submucous tissue. Ova and empty shells of the Bilharzia were present in large numbers throughout the substance of the diseased tissues, and also in the mucous and other exudations upon the surface of the lining membrane of the bladder. Deeper down lay the parasites themselves, in smooth-walled spaces, which communicated with the blood-vessels, and therefore evidently were really nothing else than altered blood-vessels. The alimentary canal of the worms was always full of blood-corpuscles, which no doubt served them for food.

How the ova effect their escape from the spaces in which the parent worms lie, and how they manage to pass through the substance of the mucous membrane does not seem to have been made out. Is it possible that the sharp spine with which each of them is provided enables them to work their way gradually outwards, under the influence of the pressure to which they are subjected by the contraction of the muscular wall of the bladder? Dr Cobbold, in discussing the purpose served by the spine, thinks that it acts as a "holdfast," giving purchase to the embryo, and so aiding it in the violent efforts which it sometimes has to make before it can get out of its shell.

In a great many cases the ureters were affected as well as the bladder; and sometimes they suffered even when it escaped; in very exceptional instances the morbid process extended even to the pelvis of the kidney. The lesions in the ureter were identical with those in the bladder, but were far more serious in their effects, inasmuch as they obstructed its channel, leading to hydronephrosis, pyelitis, and at length to complete destruction of the renal cortex. The natural result of such an affection would be the death of the patient by gradual exhaustion, and this was often observed. But in most of the instances the direct cause of the fatal issue was either pneumonia or dysentery. In regard to the latter disease it was at first a question whether it might not also be dependent on the presence of the parasite. For, besides the veins of the urinary apparatus the only other vessels in which the Bilharzia was found were the portal vein and its tributaries, including the radicles of the intestinal veins. Ova seemed, in fact, to be scattered through the coats of the bowel exactly as they were through those of the bladder; and the mucous membrane of the bowel showed similar patches of inflammation. But although it is not unlikely that symptoms identical with those of dysentery may sometimes be produced by the Bilharzia, Griesinger soon satisfied himself that the common endemic dysentery of Egypt could not be attributed to this cause. On the other hand, he was inclined to think that in some cases the morbid process set up by the Bilharzia in the urinary apparatus gave rise to acute and rapidly fatal "typhoid" symptoms. And he also held it an open question whether the contamination of the blood by the parasite might not give rise to septicæmia. That the eggs may be carried to distant parts of the blood-stream is shown by a case in which a few empty shells were found in the interior of the left chambers of the heart.

In the *treatment* of endemic hæmaturia, Dr Harley thinks that he has obtained good results from daily injections into the bladder of from twenty to thirty grains of iodide of potassium dissolved in five ounces of warm water. Dr Guillemard, however, has recorded a case in which even a weaker solution than this set up acute cystitis, and Dr Cobbold had previously expressed a strong opinion that such procedures are more likely to do harm than good. Dr Harley has also prescribed, with some apparent advantage, draughts each containing ♏xv of oil of male fern and the same quantity of oil of turpentine. When there is a tendency to the formation of uric acid calculi it should of course be counteracted by the administration of alkalies or of salts of the vegetable acids.

Malformation and Fixed Malposition of the Kidneys.—In some persons the two kidneys have their lower ends united in front of the spine so as to form a body of the shape of a horseshoe. The chief clinical importance of this condition lies in the fact that if the abdomen is thin and flaccid it may easily lead to the supposition that a morbid growth is present in the lumbar glands or that the aorta is affected with aneurysm. The occurrence of hydronephrosis or of pyonephrosis in a "horseshoe kidney" would also be very likely to be misinterpreted during life. In other individuals one kidney—generally the left one—lies at a lower level in the abdomen than natural, generally over the sacro-iliac synchondrosis, or even within the pelvic cavity. The organ may then be mistaken for a tumour of some kind, as in a case recorded by Mr Durham in the 'Guy's Hospital Reports' for 1860. Or, in the female, it may interfere with parturition.

Moveable Kidney.—In some persons, instead of the kidneys being fixed deeply in the loins, one or both of them may become loose, so as to move in various directions, downwards, forwards, or inwards, to be readily felt by the hand through the abdominal wall and to be almost as readily pushed back for the time into the natural position. At the same time, it is said, the lumbar region may be felt to be flattened or even hollowed, and percussion may yield an abnormally tympanitic note there. This condition occurs much more often on the right side than on the left. Among ninety-one cases collected by Ebstein, there were sixty-five in which the right kidney was moveable, fourteen in which the left was moveable, and twelve in which both were moveable. As a rule, the place in which the kidney is felt is in the iliac fossa, or somewhere between this and its natural seat. But it is said sometimes to "float" and to come into contact with the front wall of the abdomen, or it may be in front of the spine. One cannot conceive of a kidney as *floating* without its having peritoneum on both sides of it; and there appears to be evidence that it does actually make for itself such a covering by pushing forwards the membrane that should naturally be in contact only with its anterior surface. In vol. xxvii of the 'Pathological Transactions' may be found a case observed by Dr Goodhart, in which the right kidney, while lying on the spine and over the psoas muscle, had become completely turned over, so that its anterior surface looked backwards; both surfaces were covered by peritoneum. As a rule, however, a moveable kidney glides about behind the serous membrane, merely dragging this with it to a slight extent.

The *causes* of the affection appear generally to be complex. Sometimes, perhaps, the organ is pushed downwards by an enlarged liver; in one case that I remember to have seen in the post-mortem room of Guy's Hospital, the liver was cancerous. Cruveilhier thought that displacement of the right kidney was often an indirect result of tight lacing, through its altering the position and shape of the liver. But although a moveable kidney is much more often seen in the female sex than in the male (the proportion being as eighty-two to fourteen) it is believed to occur chiefly in women of the labouring classes who are not so likely to wear close stays. And it seems often to be due mainly to a relaxed state of the various structures forming the walls of the abdomen in consequence of frequent child-bearing. Sometimes, perhaps, an injury plays a part in determining the displacement. Thus, Ebstein cites a case by von Dusch, in which a woman who had had eleven children, and whose abdomen was very loose, fell downstairs and struck her right side; soon afterwards she felt a tumour in the right hypochondrium. Becquet has maintained that a principal cause of mobility of the kidney is congestion and swelling of the organ, recurring at the menstrual periods. The objection to this view is that under normal circumstances such congestion is not known to take place. Dr Roberts, however, in 'Reynolds' System,' alludes to two cases in which a displaced kidney seemed to become larger and more sensitive to the touch each time that the catamenia appeared. Patients in whom the kidney is moveable are often between the ages of twenty-five and forty, but Stoffen has observed this condition in children not more than nine or even six years old.

Mobility of the kidney, with consequent displacement of the organ, does not necessarily give rise to any *symptoms*. Walther, of Dresden, some years ago examined a number of persons with this object, and detected the affection in many cases in which there were no symptoms of it whatever. But, on the other hand, some persons experience a sense of weight and pressure in the abdomen, a feeling of dragging, as though something were loose in its cavity, or more or less intense pain which may radiate in various directions, to the ribs, the shoulder, the epigastrium, or the external genitalia. Sometimes there is nausea or vomiting. They may also be liable to attacks of intense suffering, attended with faintness and collapse, during which the kidney becomes exceedingly tender, and which are said to be sometimes accompanied with increased dulness on percussion, indicating that a considerable amount of inflammatory exudation is thrown out about the organ. Active exercise, whether walking or riding, often brings on or aggravates the pain; some patients are prevented by it from standing upright, or from turning round in bed, or even from lying on one side. There is often an apparently disproportionate degree of anxiety and of depression of spirits, amounting to hypochondriasis.

The *diagnosis* of a moveable kidney is sometimes easy, sometimes difficult and uncertain. The affection may have to be distinguished in different cases from a fæcal accumulation, from an enlarged spleen, from a distended gall-bladder, from a mass of swollen glands, or from an ovarian cyst with a long pedicle. Ebstein mentions an instance in which a hydatid cyst in the mesentery was mistaken for it; and this I can well believe, for in a case which I myself saw, and in which there were several such cysts, some of them were oval in shape, and of almost exactly the size of the kidney.

The *treatment* of this affection generally resolves itself into keeping the patient in bed when there is severe pain, and applying fomentations and poultices. Afterwards an elastic abdominal belt should be applied, having a concave pad so placed as to maintain the kidney in its proper position. It is often, however, very difficult so to adjust an apparatus as to effect this object, and not to do more harm than good.

When a moveable kidney is attended with unbearable suffering, it is doubtless justifiable to extirpate the organ. According to Czerny ('Trans. Internat. Congress 1881') this has been done in twelve cases. Four of them ended fatally, three by peritonitis, one in consequence of the opposite kidney being diseased; the others were completely successful, the patients not only recovering from the operation, but being also cured of the symptoms on account of which it was undertaken. No fewer than seven of these twelve cases of nephrectomy occurred in the practice of a single surgeon, Dr Martin, of Berlin.

More recently Hahn has proposed to cut down upon the kidney by a lumbar incision, and to fix it in its proper place by sutures. Several cases are recorded by Ceccherelli ('Revista Clinica,' April, 1884), quoted by Sir Spencer Wells.

Another suggestion made by Czerny is to inject alcohol into the connective tissue around the organ, while the patient is maintained in a recumbent position.

ADDISON'S DISEASE

The anatomical changes in the adrenal bodies—The symptoms: melanoderma, asthenia, cardiac failure, gastric disturbance—Order of the symptoms—Course, mode of death, and duration of the disease—Controversial points—Origin—Relation to tubercle—Theory of the symptoms—Treatment.

Among the most remarkable of all the diseases with which modern pathological anatomy has made us acquainted is one which has its seat in the suprarenal bodies—almost the only substantive disease to which these organs appear to be liable. It was first recognised clinically by Dr Addison (some years before the publication of his work on the subject in 1855), and consequently it is very often spoken of as "Addison's disease." The symptoms which characterise it may be briefly enumerated as —(1) a gradually increasing and fatal debility or asthenia; (2) a peculiar discolouration of the skin; (3) a liability to nausea and vomiting.

Anatomy.—The exact characters of the affection of the suprarenal bodies which gives rise to these symptoms vary considerably in different cases, but only within such limits as may fairly be supposed to belong to the successive periods of a single morbid process. The earliest change seems to be the formation, within the medullary substance, of a firm grey or whitish mass, which is more or less nodulated at its growing edge and sometimes surrounded by clusters of what appear to be miliary tubercles. In this stage, however, the disease rarely becomes the subject of anatomical investigation. When death has occurred, the grey material is generally found to have already extended into and destroyed the cortical substance of the organ. It is then greatly enlarged, hard, and of irregular form; when a section is made of it the cut surface shows no trace of the natural structure; it may be of a more or less uniform grey or greenish-grey colour, which, according to Dr Greenhow ('Croonian Lectures,' 1875), quickly acquires a pink hue when exposed to the air. In most cases, however, certain parts of the grey substance have undergone caseation, forming rounded yellow nodules embedded in it; and, at a still later stage, the conversion is complete, so that there is only a single large yellow mass. Softening often takes place at this period, and a creamy liquid results, which used formerly to be mistaken for pus. Finally, a process of absorption begins, and the diseased organ, from being many times larger than natural, shrinks into a very small puckered mass, in which irregular nodules of calcareous matter are deposited.

During the early stages of this process the fibrous envelope of the organ becomes swollen until it is from half a line to two lines in thickness. Adhesions to the nighbouring parts are also formed, principally to the diaphragm, liver, kidney, pancreas, vena cava, or stomach. But in the later periods of the disease the capsule of the suprarenal body is no more to be recognised than the structure of the organ itself. I once examined a case in which nothing was left but a hard puckered fibrous knot, sending out bands into the fatty tissue in which it lay embedded, and having interspersed within it a number of irregular calcareous masses.

It is a question whether Addison's disease is ever limited to a single suprarenal body. Some cases have been recorded in which one has been said to have been healthy. But I am not sure whether any of them would bear critical inquiry. I believe that all those who have worked specially at the subject have invariably found both organs affected. One of them, however, is generally attacked earlier than the other, and on post-mortem examination it may be found to have undergone complete destruction, while its fellow is still in a comparatively early stage of the disease.

The new tissue which is formed at the commencement of the morbid process consists of small round cells of lymphoid character lying in the meshes of a delicate, wavy, fibrillated stroma. These cells may subsequently undergo development, and form a well-marked fibrous tissue, for I have found some parts, which were still grey and translucent, to consist entirely of such tissue, with some elongated cells and oval nuclei interspersed between its fibres. It seems, from Dr Wilks's description, that the specimens that he examined were generally in this stage. He speaks of a material, which he says is "without structure, or sometimes slightly fibrillated, or containing a few abortive nuclei or cells." When caseation has taken place the microscopical appearances are the same as those of any other structure which has undergone this change.

Melasma.—The discolouration of the skin which occurs in Addison's disease varies considerably in different cases, not only in intensity, but also in tint. Thus it is variously described as being yellow brown or greenish brown, dusky, smoky, or as if stained by walnut juice. The term "bronzed-skin" was at one time commonly used for it, but is not a very apt one. One can hardly give a better idea of the hue than by saying that it resembles that of one or other of the dark races of mankind. As in persons who have long resided in hot countries, the discolouration is deeper on the face and neck, and on the backs of the hands, than on the covered parts of the body generally; but it is very marked on the genital organs and about the pubes, and also in the axillæ, navel, nipples and areolæ. In extreme cases it may be universal, so that the patient looks almost like a negro; but I believe that it is never uniformly deep over all parts of the body. There is no sharp line of demarcation between the discoloured parts and those which remain pale; they shade off into one another.

In most cases, some parts of the surface also present a few small black spots, resembling minute pigmented moles; these have comparatively defined outlines. Dr Greenhow has laid especial stress upon them.

Parts of the body which are habitually rubbed are apt to become the seats of pigment. Thus a brown ring is often seen round each leg where the garters have pressed; or round the waist in women, where the petticoat strings have been tied. A case is quoted of a baker's lad, whose shoulders showed dark stripes corresponding with the bands by which his basket had been slung over his back. So, again, the application of a blister to any part of the skin is followed by the formation of a brown patch; and deep stains are left by eruptions which had been attended with much congestion of the skin.

The stains and patches just alluded to are but an exaggeration of pigmentations that occur in less marked forms even in healthy persons, particularly in those of dark complexion. And the diffused discolouration of the face, neck, and hands may be compared with what would naturally be observed in anyone exposed to the heat of a tropical sun; it may fairly be supposed that the pigment is really deposited beneath the influence of solar rays, which would not be sufficiently intense to produce such an effect in a healthy individual. It is probable that if a patient affected with Addison's disease could be kept in the dark, or even be prevented from going into the sunlight, the exposed parts of his skin would remain pale. Some years ago, Dr Pavy had under his care in Guy's Hospital a woman who suffered from the characteristic constitutional symptoms; she had on her legs reticulated markings, such as are seen in persons who habitually sit before the fire, and which commonly receive the name of *ephelis ab igne.* She declared, however, that her limbs had not been specially exposed to the influence of heat. A short time afterwards she died, and disease of the suprarenal bodies was found to be the cause of her death. I am disposed to think that the markings on her legs were really developed under the influence of that disease, affording another example of its tendency to lead to the exaggeration of pigmentation that may also occur independently of it. I must, however, add that one not infrequently sees ephelis ab igne in persons in whom one cannot ascertain that the lower limbs have been exposed to the heat of a fire, but in whom there is no reason to doubt that the suprarenal bodies are healthy.

Again, the discolouration of the genital organs, axillæ, and nipples may also be regarded as an intensification of physiological deposits of pigment; and the small black scattered spots described by Dr Greenhow are perhaps really representatives of the moles which they resemble. The same may also be said of certain marks which are seen on mucous membranes. Thus each lip commonly shows a bluish-black streak along the line of contact with the other lip; and on the buccal mucous membrane irregular and ill-defined brownish stains may occasionally be seen. These last Dr Greenhow has since traced to the irritation set up by the pressure of protruding teeth. But probably they may also occur independently of any such cause, for similar stains are found in the lining of the cheeks of Lascars. Another part of the mucous membrane upon which Dr Greenhow has noticed stains in Addison's disease, is the side of the tongue; they have been of a purplish blue, or inky hue, and were always near the free margin. The conjunctivæ always remain pearly white, contrasting with the dark colour of the surrounding parts of the face.

The microscopical appearances of the discoloured parts of the skin bear a close resemblance to those which may be observed in individuals belonging to the darker races of mankind. The pigment consists of yellowish-brown granules, and its chief seat is in the deepest layers of the rete mucosum, close to the papillæ.

Dr Greenhow, however, remarks that he has sometimes found traces of pigment in some of the more superficial scales of the epidermis, and likewise in the cutis. German observers also have detected pigmented connective-tissue cells within the papillary layer; but there is not anything really unusual in the presence of colouring matter in this situation, for it occurs in many pigmentary moles.

It is still a question whether any of the internal organs are ever discoloured in cases of disease of the suprarenal bodies. Addison's work contains drawings of a mesentery, intestine, and omentum, over all of which numerous minute black spots were scattered. They were taken from a case which he believed to be one of that disease; but it appears doubtful whether that view was correct, and also whether the pigmentation of the peritoneum may not have been due to a former attack of peritonitis.*

Asthenia, &c.—Next to the discolouration of the skin, progressive weakness is the most striking and important of the symptoms of Addison's disease. The patient becomes more and more languid as it advances. He takes to his bed, and his prostration increases until he becomes unable even to sit up. Merely being raised into a sitting posture may be followed by faintness and giddiness. Yet there is often but little loss of flesh; and after death a certain amount of fat may remain beneath the integuments and about the viscera. Anæmia also, though usually present, is not extreme.

The heart's action is remarkably weak, and the *pulse* very small and compressible. Breathlessness, palpitation after any muscular effort, frequent sighing or yawning, and persistent hiccough sometimes occur; and the patient may complain of more or less pain in the loins or in the epigastrium.

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* [Dr Carrington has lately observed the normal pigmentation of the pia mater covering the bulb much intensified in a case of Addison's disease.—Ed.]

The symptoms of *nausea*, with retching and vomiting are very rarely wanting.

Course.—The development of the symptoms of Addison's disease is generally gradual; but their order is variable. Sometimes the skin becomes dark a long while before the general health begins to fail. Thus, a young lady, whose case was recorded by Addison, had had the "bronzing" for one year before her death, but appeared ill during only about four months. A man who was under the care of Mr Harris, of Hackney, had been noticed by his wife to be getting darker for two years, but mentioned no other symptoms as having been present for more than six months. In the great majority of cases, however, the patient suffers from progressive asthenia for a considerable period before pigmentation of the skin becomes noticeable, and if the former should develop itself rapidly in a severe form, death may occur at a time when the latter is still entirely absent.

In a series of 228 cases, collected by Dr Greenhow, there were twenty-nine in which, when the patients died, the skin was either not bronzed at all, or to a scarcely appreciable extent. But in eighteen of these some other disease was likewise present, which probably was immediately concerned in bringing about the fatal issue. In each of the remaining eleven cases in which there was no such complication, the patient's illness had been of comparatively short duration; in one only had it lasted eight months, and in another four months. However, at Guy's Hospital one case has occurred in which there was no discolouration, but in which the patient had been ailing for twelve months before his death. Probably the most precise statement which we are justified in making is that the skin is always dark when the constitutional symptoms of Addison's disease have lasted more than a year.

Although the course of Addison's disease is progressive, its rate of progress is by no means uniform. Dr Greenhow has pointed out that alternate exacerbations and remissions occur, which can only partially be traced to changes in the conditions under which the patient is placed. It is even said that the depth of discolouration of the skin may alternately diminish and increase.

Death sometimes takes place very gradually. The mind may be clear to the last, or the patient may lie in a drowsy and semicomatose state, from which, however, he may be roused to give pertinent though slow answers. In such cases the temperature falls considerably below normal. Or death may be preceded by muttering delirium or coma. One of Dr Greenhow's patients had a convulsive fit, and lay for hours with closed jaws and rigid limbs; whenever he was touched convulsive twitchings took place. In some cases, again, the fatal termination occurs unexpectedly, the patient having apparently been in a fair state of health a few hours before. Thus a young man some years ago was under the care of Dr Wilks for eczema which was getting better, when he was attacked with diarrhœa and vomiting; he became exceedingly prostrate, and died in less than twenty-four hours. Disease of the suprarenal bodies had never been suspected, but was found to be the cause of his death. Quite recently a patient of Dr Pye-Smith's who was in the hospital with Addison's disease, but was about to leave, became suddenly worse and died in a few hours.

The *duration* of Addison's disease appears to be very variable. There are at any rate great differences in the length of time which elapses between the commencement of the symptoms and the death of the patient; but it must be admitted that the period at which the affection of the suprarenal bodies begins to develop itself cannot be determined. Dr Wilks some years ago stated that the average duration of the cases which he had collected was eighteen months. One of the most rapidly fatal instances is that of a girl who came under Dr Greenhow's care, and who, although weak and supposed to be sunburnt, attended school until about a week before she died. The longest case that I have met with was one in which I made a post-mortem examination in 1865. The disease had been diagnosed by Dr Gull at the time when the patient was first admitted into the hospital in 1860; he then said that his skin had already been dark for two years. In this instance, therefore, the disease lasted for at least seven years.

Spurious and aberrant cases.—In describing the morbid anatomy of Addison's disease, I have implied that the affection is always of one kind. This, however, was not the opinion of Addison himself, who, in his original work, included four cases of cancer of the suprarenal capsules, and who at that time thought that any affection completely destroying the organs would be capable of giving rise to the characteristic symptoms. It was, I believe, Dr Wilks who first pointed out that in all genuine instances one particular morbid change in the capsules is found. He showed that in Addison's cancerous cases the proper symptoms were not really present. He also first defined the characters of the discolouration of the skin. The errors into which Addison fell in regard to these points are much to be regretted, for they have led to much misunderstanding and even to doubts as to the existence of the disease which have not yet been completely dispelled. I should, indeed, have supposed that this was proved, beyond the possibility of dispute, by the mere number of cases that have been reported, many of which have been diagnosed during life in the most positive manner. In his lectures delivered before the Royal College of Physicians in 1875, Dr Greenhow was able to cite 183 recorded cases, in every one of which the special form of discolouration of the skin, and some at least of the constitutional symptoms were present.

The cases which have been supposed to lead to conclusions adverse to those maintained by Dr Wilks and other modern supporters of Addison's views fall into two groups.

In the first group comes those in which the capsules have been diseased *without giving rise to the characteristic symptoms.* We have seen that in some uncomplicated cases of Addison's disease death has occurred before the time at which bronzing of the skin necessarily develops itself; and in others a rapidly fatal issue has been due to some other malady, such as phthisis or Bright's disease, by which the constitutional symptoms of Addison's disease were masked. Now, apart from such cases, Dr Greenhow has been able to state that in every recorded instance of suprarenal disease without symptoms the affection was really different from that which alone is known to be capable of causing symptoms. And in most instances it was cancer. The suprarenal bodies, indeed, are liable to be the seat of primary malignant growths, as well as to certain secondary nodules of various kinds. I remember one case in which each of them was three or four times as big as the kidney, so that there was a large tumour observable during the life of the patient. It may seem strange that such an affection should not cause the symptoms of Addison's disease. But, as Dr Moxon has pointed out, there is an analogous circumstance in the fact that the most extensive cancerous growths in the liver often fail to produce jaundice. Indeed, there is reason to believe that the cause of the symptoms in question is the extension of morbid changes beyond the suprarenal bodies to the semilunar ganglia and sympathetic nerves; and it may be that these structures are not affected in the same way by even the largest malignant tumours.*

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* [Cases of Addison's disease have, however, been recorded by excellent observers in which, instead of the usual changes, simple atrophy of the adrenals has been found: by

The second group of cases which have been supposed to justify doubts as to the value of Addison's discovery consists of those in which bronzing of the skin has been said to exist *without the characteristic suprarenal affection.* But it must be borne in mind that discolourations of the skin are from time to time met with, which may be mistaken for that of Addison's disease by an unpractised eye, although they really differ altogether from it. In one of the most curious of these the skin becomes, as it were, *piebald.* Some parts are much darker than natural, others are entirely devoid of pigment, and the hairs upon them white. The white areæ always have very definite convex borders, and thus look as if they were encroaching upon the bronzed parts, which, on the other hand, shade off very gradually into the healthy skin. Thus the absence of pigment in some places is far more obvious than its excess in other places; and the affection is accordingly now known by the name of Leucoderma (λευκός = white). Addison, however, himself confounded it with that which occurs when the suprarenal capsules are diseased; and in one of his cases which appears to me to have been a genuine one the skin did actually present an affection of this kind. All subsequent experience, however, has tended to show that leucoderma is altogether distinct from the discolouration which occurs in Addison's disease. I have never heard of any other instance of it associated with any impairment of the general health. But not long ago a case of this kind was sent up to Guy's Hospital from a distant county as one of bronzed skin.

Again, as Dr Greenhow points out, elderly persons of indigent circumstances and unclean habits, especially when infested with vermin, often have the skin of the back, chest, and abdomen deeply pigmented, so that the presence of disease of the suprarenal capsules might be suspected. The roughness of the cuticle in such cases affords a distinction. Dr Greenhow relates two or three cases of chronic phthisis in which a pigmentation was present, somewhat resembling that of Addison's disease. The chloasma of women pregnant or affected with uterine disease, the discolouration of skin produced by malarious and tropical fevers, or by liver diseases, and even tinea-versicolor, have each been mistaken for the bronzed skin of suprarenal disease. But the most extraordinary instance of perversity is perhaps that afforded ('Virchow's Archiv,' 1870) by a German observer, who met with a case of scleroderma, in which large patches of the skin in succession became deeply pigmented, sometimes in a single night. The patient, an old woman, had Bright's disease, and died of pneumonia. Her suprarenal capsules were healthy, but her medical attendant, instead of seeing that his idea of the case was altogether wrong, proceeded to base upon it an entirely new theory with regard to Addison's disease, ascribing it to a functional disturbance of the cerebro-spinal system. The truth is that one cannot accept unreservedly the diagnosis of Addison's disease at the hands of a medical man who has not already seen other cases, and so made himself particularly acquainted with its characters. And even in a large hospital the disease is not common enough to come under the observation of every student.

Ætiology.—Addison's disease occurs much more often in males than in females. According to Dr Greenhow the proportion is as 119 to 64. The age of the patient is generally between twenty and fifty, but instances have been met with in children of five, eleven, and thirteen years of age respectively.

Dr Greenhow thinks that men engaged in hard bodily labour are especially liable to this disease. He even thinks that its starting-point is sometimes a direct strain of the back or even a blow on it. He mentions seven such cases; one is that of a woman who constantly asserted that she

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Legg ('St Barth. Hosp. Rep.,' 1874), Davy ('Path. Trans.,' xxxiii, 360), Goodhart (*ibid.*, p. 340), and B. Fenwick (*ibid.*, p. 354, with table of cases). See also the same 'Transactions' for 1885, and the 'Clin. Trans.,' vol. xix.—Ed.]

had never recovered from a strain in the back, which had occurred while she was turning a mangle some years before; another patient had always had ill-health from the time when she fell downstairs; a third dated his illness from a fall through a trapdoor.

In none of these instances was there any discoverable affection of the bones or ligaments of the spine. But in many other cases vertebral disease has been present. Dr Greenhow mentions eighteen instances of this; and others, hitherto unpublished, have occurred at Guy's Hospital. The lower dorsal or upper lumbar vertebræ have generally been the seat of the mischief, and there have always been abscesses in or near the psoæ muscles. Very often a sinus has led from the abscess to the neighbourhood of the adrenals; or it has at least been clear that the disease spread continuously from one structure to the other. In some cases the vertebral disease was directly traceable to injury, in others it appeared to be of strumous origin. Indeed, even when its immediate cause was a blow or fall, a constitutional taint most probably also played a part in its causation.

Nature of the change.—This brings us to the important question whether the affection of the suprarenal body is inflammatory or tubercular. Addison spoke of it as scrofulous, but formerly this name was applied to all caseous masses. Wilks was inclined to deny that the disease was of a tubercular nature; he maintained that cases in which well-marked tubercles were discovered in other viscera were in fact exceptional. Virchow and Rindfleisch, however, term Addison's disease a tuberculosis. They describe as the earliest stage of the affection one in which nothing but a cluster of grey nodules exists in the medullary substance. I have myself observed the same thing, but only in cases of general or at least widely-diffused tubercular disease—in which bronzing of the skin was absent or but doubtfully present; it would be very difficult to establish any connection between such cases and those of Addison's disease. I myself, however, believe that the affection is of a tuberculous nature. In the first volume of this work (p. 79) I pointed out that tuberculous diseases occur in at least four or five distinct forms, each more or less limited to one particular group of organs, but all of them in individual cases so frequently connected with one another as to show that their essential nature is the same. And, in cases of adrenal disease, whatever morbid changes are found in the lungs or in other parts of the body bear unmistakable marks of a tubercular origin.

Pathology.—What relation can be discovered between the morbid change and the symptoms that characterise the disease? I know of no tenable hypothesis unless it be that the ganglia and branches of the sympathetic nerve, which are so intimately connected with the adrenal bodies, are involved in an inflammatory process, starting from them. Dr Habershon in 1863 dissected out the semilunar ganglia, and found that they and their branches of nerve were surrounded on the side on which the suprarenal body was more severely affected by dense fibrous tissue in excessive quantity. Similar observations have since been made by other pathologists both in England and abroad. The semilunar ganglia have been found enlarged and reddened; and under the microscope their cells have appeared opaque and granular, and remains of hæmorrhages into their substance have been discovered. The fibres of nerve-trunks embedded in adhesions have also been shown to be in a state of fatty degeneration. [Tuckwell, 'Path Trans.,' 1868.] Again, one or two cases have been recorded to prove that the several symptoms of Addison's disease may be caused by affection of the semilunar ganglia independently of any primary change in the suprarenal bodies. Sir William Jenner, when President of the Pathological Society, mentioned such an instance as having come under his observation; and as far back as 1847 Dr Bell Fletcher recorded a case. [Also Barlow and Coupland, 'Path. Trans.,' 1885.]

What we know of collapse as a result of a sudden shock to the semilunar ganglia accords with the opinion that the extreme debility of Addison's disease may depend upon chronic changes in these important nervous centres; and the nausea and vomiting are still more readily accounted for.

But no such obvious explanation suggests itself of the peculiar pigmentation of the skin. This has been attributed by Jaccoud to irritation of the vaso-motor nerves; but Risel, who has carefully discussed the question, concludes that some blood-change must also occur. It seems to me that the solution of this difficult problem may perhaps involve the principle, which I have already laid down, that the "bronzed skin" of Addison's disease always corresponds with some form of pigmentation that may occur in health under the influence of stimuli, or in one of the dark races of mankind. It is, I think, probable that under normal conditions this, like other organic processes, is kept in check by the sympathetic nerves. And if in disease their controlling influence is withdrawn, we cannot be surprised that pigment should be laid down in excess, or at least in quantity altogether disproportionate to the intensity of its exciting cause. Such an explanation, indeed, involves more than one hypothesis, and it may appear too far fetched. But I doubt whether any simpler one will account satisfactorily for the facts.

Treatment and prognosis.—The treatment of Addison's disease may unfortunately be summed up in a very few words. The vomiting must be combated by appropriate medicines; but these are too often altogether ineffectual. For the debility and prostration tonics and stimulants are obviously indicated; but they likewise generally fail to do any good. It occurs to me that the use of iodine (whether internally or as an application to the lumbar regions) might possibly lead to the subsidence of those inflammatory changes which have been shown to occur in the connective tissue round the semilunar ganglia and branches of sympathetic nerve; and if these changes should be the cause of the symptoms, such treatment might do good. Hitherto, so far as I am aware, no positive proof has been given that recovery from this disease ever takes place. But it is certain that many patients, after having been kept in a hospital for a long time, and perhaps after having been admitted over and over again, have been lost sight of. This might well have happened in the case which I have already mentioned, of a young man in whom Addison's disease was diagnosed in Guy's Hospital five years before the time he died. I have already briefly described the morbid appearances which were found in the organs, and which seemed to show that they had been destroyed for a considerable length of time. And, since the degree to which the sympathetic centres and nerves are involved no doubt varies in different cases, there is little difficulty in admitting that recovery may sometimes take place even after the complete development of the changes in the suprarenal bodies which constitute the primary pathological basis of the disease.

Other Affections of the Adrenals.—[These organs may be the seat of miliary tubercles in cases of general tuberculosis; or of hæmorrhage; or of cancer, either by contiguity from the kidney, lumbar glands or vertebræ, or as a secondary deposit; or of lardaceous transformation; or of embolism; or of secondary pyæmic abscess. Lastly, they may apparently become atrophied without preceding enlargement. But none of these pathological states are of clinical significance. In a textbook of medicine, Addison's disease is the only lesion of the adrenals which demands a separate place.]

GENERAL DISEASES AFFECTING THE JOINTS

GOUT

Nomenclature—Onset and symptoms of a first attack—Subsequent course—Tophi—Pathology of gout—Lithæmia—Ætiology—Diagnosis—Prognosis—Gouty kidneys—Treatment.

Unlike so many other diseases which seem to have escaped the notice of the ancient writers, gout was well known to them. Hippocrates gave an account of it. There is a scene of Lucian's, in which *Podagra* is one of the dramatis personæ.

In modern times, we find an essay on gout by Sydenham, who was very competent to write about this disease, having himself suffered from it for thirty-four years. His masterly description was copied by Cullen, and it has apparently formed the basis of almost all that has since been written upon the subject. The name which Sydenham employed for gout was the classical one of Podagra; but this is evidently unsuitable, for the foot is not the only part affected. Other writers have called it arthritis; but to that term also there are obvious objections, since it is applicable to, and has in fact been used for, other inflammatory affections of the joints. By far the best word is Gout, which has never been applied to any disease but the one now under consideration, and which moreover is closely allied to the names given to it in several other European languages. In German it is Gicht; in French Goutte. The derivation of these words is believed to be from the Latin *gutta*, the notion being that of a morbid humour *dropping* into the joints.

The gouty fit.—In a large proportion of cases, when gout occurs for the first time in a young subject, it begins in a very curious way. The patient is asleep in bed, when he is awakened, about two o'clock, or between two and five, by a pain in one of his feet, generally in the metatarso-phalangeal joint (or "ball") of the great toe, but sometimes in the heel, ankle, or instep. This pain is described by Sydenham as being like that of a dislocation; and yet, he says, the parts feel as if tepid water were poured over them. Then follow chills and rigors and a little fever. The pain, which was at first moderate, becomes more intense. It is characterised as a grinding, crushing, wrenching pain; or as a burning, such as would be caused by a hot iron pressed into the joint. The patient keeps changing the position of his foot, in the vain hope of finding a place in which it may lie in comfort. He cannot bear the bedclothes to touch it. The least vibration of the floor causes him extreme distress, so that those about him have to tread the room with the lightest possible steps, and the passage of a waggon along the street below almost sends him mad with rage. For, as Sydenham puts it, "a fit of gout is a fit of bad temper."

Towards morning the patient has a sudden and slight respite, which he wrongly supposes to result from his having at last found a comfortable position. He perspires gently and falls asleep. He wakes freer from pain, and then finds that the part is swollen. Till then, the only visible swelling had been that of the veins round the painful joints. For the next two or three days the pain becomes worse towards evening, and abates in the morning. Then, generally, the other foot begins to swell, and pains are felt in it, like those in the one which was first attacked.

A joint affected with gout, besides being painful and swollen, is also of a deep red colour, tense, and shining. As soon as the patient can bear the pressure of the finger, one finds that the skin over it pits, or (in other words) that there is œdema. Subsequently the cuticle peels off; and the part then itches very much.

The amount of febrile disturbance is proportionate to that of the local inflammation. This is a point of some consequence, as it constitutes one of the distinctions between gout and acute rheumatism; in the latter disease there may be high fever, with scarcely any joint affection.

In robust patients, who have not had more than a few previous attacks, the duration of a fit of the gout is about a fortnight. But in persons of advanced age and broken-down constitutions it may last two months or even longer. In that case, however, the length of the attack is rather apparent than real; for it is in fact made up of a series of irregular minor fits, which gradually become milder.

It is generally said that before a first seizure of gout the patient appears to be in his usual health. Certainly he seldom has any idea of what is about to befall him. Sydenham, however, observes that indigestion and flatulence precede the attack; and Dr Garrod says that the premonitory symptoms are sometimes very distressing. Trousseau remarks that in addition to symptoms of hepatic disorder the patient often has an irregular and capricious appetite, prefering acids and meats strongly spiced, as if he felt the necessity of stimulating his torpid organs of digestion.

After an attack of gout, on the other hand, a man often feels much better than he had done for some time before. He is more active, and free from many uncomfortable feelings that had before troubled him. Sooner or later, however, these return, and are the prelude to a second seizure. Sometimes this does not occur for two or three years after the first; but more often the interval is not more than a twelvemonth, and it may be even less. The second attack is in its turn succeeded by others, and always at shorter intervals; until, at last, the patient may be scarcely ever free from the disease.

Distribution.—It has already been stated that the ball of the great toe is the joint most frequently first attacked by gout. Sir Charles Scudamore found that this joint was affected on one side or the other in 373 out of 512 first seizures. When any other part shows the earliest manifestation of the disease, the reason appears generally to be that it has in some way been injured. Thus according to Dr Garrod, the knee may be attacked before any other joint, if the patient has injured it in falling from his horse, or in some other way; and a considerable time may pass after the accident before the result follows.

Whatever may be the part first affected, other joints afterwards suffer in almost all cases. In the very first seizure, two or three different articulations may be attacked. These are generally some of the small joints, such as those of the toes or fingers, the ankles or wrists. Even in the most advanced and intractable cases the largest joints of all—the shoulders and hip-joints—are comparatively seldom involved.

Anatomy.—When a patient has suffered from repeated attacks of gout, the affected joints become greatly deformed. This is generally much more marked in the fingers than in the toes, no doubt because their natural movements are so much more free. The fingers in fact become bent in all directions; sometimes inwards, sometimes outwards. A very common state is for the metacarpo-phalangeal and the second phalangeal joints of a finger to be stiffly flexed, while between them the first phalangeal joint is over-extended, its knuckle being represented by a deep hollow.

Sometimes the sides of the fingers are greatly enlarged. Sydenham compares their appearance to that of a bunch of parsnips. But in many cases they are very little altered in form.

Another morbid appearance, which is commonly present in long-standing cases of gout, consists in the presence, in the tissues round the joints, of masses of a white material having the consistence of putty or mortar, or hard and dry like chalk. These masses are called *tophi*, or popularly "chalk-stones." They do not, however, contain salts of lime in any great quantity, but consist mainly of urate of soda. This salt is also found deposited in the articular cartilages of the affected joints. It there looks as if it were upon the surface of the cartilages, covering them more or less completely, and appearing just as if it had been laid on by a brush. However, on making a section of the cartilage, one finds that the deposit is really in its substance. Examined by the microscope, it proves to consist entirely of crystals These are bundles of very fine needles, the centres of which (according to Cornil and Ranvier) commonly correspond with the cells of the cartilage. It is in the superficial part of the cartilage that the crystals are most densely crowded; they often make that part quite opaque to transmitted light. Towards the articular end of the bone they are more thinly scattered; here they often traverse the whole thickness of a cartilage cell, which thus looks as if it were impaled by them. The synovial membrane may contain similar deposits of the urate; but they are much more apt to occur in the fibrous structures outside joints, particularly the surrounding ligaments and tendons.

There are some discrepancies in the statements of writers as to the deposition of urate of soda in the bones. Dr Garrod says that he has never been able to find it in the osseous substance. But Cornil and Ranvier teach that it is not uncommon in the cancellous tissue of the ends of bones. They mention one case in which they observed it, and in which the bones forming the metatarso-phalangeal joint of the great toe had entirely lost their cartilages and were ankylosed together. Absorption of the articular cartilages is, they say, a common remote effect of the deposition of urate of soda in a joint.

The same salt is also deposited in many cases, even at a distance from the joints. I remember seeing a man who had a number of small ulcers, some of them in the middle of his thighs and legs, from which masses of urate of soda were discharged. The integuments of the limbs, however, very rarely present such deposits, except over articulations. But there is one particular region where they are very often to be found. This is in the external ear, and generally in or near the helix. Dr Garrod remarks, and I can confirm the statement, that these deposits in the pinna of the ear are at first fluid, the skin over them forming a vesicle of a milk-white appearance. He says that some months elapse before they become the white hard bead-like masses which are commonly seen. I am not aware that in the joints, or in any other parts except the ears, any fluid is present when the deposition of the urate of soda first occurs.

The bursæ have still to be mentioned as parts which are very liable to receive accumulations of urate of soda in gout. This is particularly the case with those over the olecranon, which may become enlarged until they are almost as big as oranges.

Sometimes, when these deposits of urate of soda are firm and white, the skin over them wears through. Nodules in the ears may in this way be cast off, and the patient be freed from their presence. Sir Thomas Watson refers to the case of a gentleman who had exposed tophi on his fingers, and who used, when playing at cards, to chalk or score the game upon the table with his gouty knuckles.

Another change to which tophi are liable is the occurrence of suppuration in the tissues round them. And in connection with this subject it may be remarked that although a joint affected with acute gout often looks so inflamed that one might almost imagine it to be ready to point, yet as a matter of fact this never happens, nor is pus ever formed. But, on the other hand, abscesses round the extra-articular deposits of urate of soda in chronic gout are very common, and the salt is freely discharged from them, mixed with pus. Dr Garrod speaks of as many as five or six abscesses of this kind being open at one time on each hand, and others on the feet, and he remarks that they give rise to very little constitutional disturbance.

Hitherto I have merely spoken of deposits of urate of soda as occurring in advanced cases of gout, and until recently it was universally supposed that they were to be found only in such cases, and, indeed, that their presence was somewhat exceptional. Sir Charles Scudamore asserted that not one gouty case in ten exhibited them. This, however, is true only so far as concerns masses of the urate deposited outside the articulations, and large enough to be seen and felt. There is reason to believe that within every joint affected by the disease urate of soda is always present in greater or less quantity. Dr Garrod mentions the cases of two patients of whom one had only a single attack of gout thirteen years before his death, while the other had had two attacks within the last two years of his life. In each case a small quantity of urate of soda was found as a white deposit upon the surface of the joints which had been affected by the disease.

Pathology.—This will be the most convenient place for the consideration of the *theory* of gout, and I must start from the fact that the blood in this disease contains an excess of uric acid; in other words, that gout is one of the manifestations of lithæmia (p. 254). Now, Dr Garrod has not only shown that uric acid can be detected in the blood in gout by an elaborate chemical analysis, but also that for clinical purposes a very simple method of determining its presence may be employed with success. About two drachms of the serum of blood are put into a flat glass dish, somewhat larger than a watch-glass, and acetic acid is added so as to give a slight acid reaction. A fibre from a piece of linen cloth is then placed in the fluid, and the dish is set aside until its contents have acquired a gelatinous consistence by evaporation. If the blood contains uric acid in excessive quantity it is deposited upon the fibre, and this becomes studded with crystals, the characteristic appearance of which can readily be identified with a pocket lens.

The serum from a blister will also yield crystals of uric acid when examined in this way, but, according to Dr Garrod, only when the blister is placed at a distance from a joint affected at the time with gouty inflammation. He states that the acid cannot be detected in the fluid from a blister in the neighbourhood of a gouty articulation, and he infers that gouty inflammation causes a local destruction of uric acid. However this may be, it appears to be certain that an attack of gout has in some way the effect of clearing the blood from its impregnation with the acid. In patients who had partially recovered from an acute seizure a very marked decrease in its quantity was found by Dr Garrod, and, indeed, in the intervals between the early attacks of gout he failed to detect any appreciable amount of it in the blood. This tendency, which gouty inflammation possesses, of freeing the blood from the uric acid which had accumulated in it, accounts for a fact which appears to be well established, and which certainly needs explanation, namely, that an attack of acute gout often leads to the rapid disappearance of certain other symptoms from which the patient had before been suffering. The symptoms in question are those which are commonly described as the effects of "irregular" gout, the ordinary affection of the joints being, on the other hand, spoken of as "regular" gout. Other epithets sometimes used instead of "irregular" are "atonic," "latent," "lurking," "masked," and "misplaced." Now, I have already described the various symptoms to which I am referring as effects of lithæmia (p. 254). It appears to me that this is at once the most simple and the most correct designation, and that we may disuse altogether the various other terms that I have just enumerated. Dr Garrod has demonstrated by direct experiment the fact that uric acid was present in the blood "in some cases where symptoms of irregular gout were present without any accompanying joint disease."

The next question is, what is the distinction between a state of lithæmia and one of gout? And to this question also one may find a satisfactory answer in Dr Garrod's views. As I have already stated, he has shown that urate of soda is invariably present in a gouty joint. This, I believe, was an original idea on his part. Still more original was the idea, which also he first suggested, that the salt is deposited, not as the result of the inflammation, but before it occurs—that, in fact, the presence of urate of soda in an articulation is a condition antecedent to an attack of gout, and very probably its cause.

In the pinna of the ear, in fact, gouty concretions commonly form without any indications of previous inflammatory action. In some cases the patient experiences sensations of heat and pricking and the part is tender, but more often he is quite unconscious of the fact that such concretions in the pinna are present. Judging from the observations which have been made by Dr Moxon and myself, in the post-mortem room of Guy's Hospital, I should be inclined to infer that in the interior of joints also the deposition of urate of soda sometimes takes place no less slowly and with the same absence of symptoms. At any rate, we have found the articular cartilages of the great toe joints encrusted with the salt in many cases in which no mention of gout had been made during life. And I should be disposed to think that the occurrence of a definite gouty seizure is often more or less of an accident in the course of an essentially chronic change.

This, however, is not exactly the view maintained by Dr Garrod. He states that among a large number of bodies of persons who are not known to have gout there were only two in which even the slightest traces of urate of soda were found in the great toe joints. And he supposes that the deposition of the salt in a joint is always the immediate precursor of a gouty seizure.

There are two conditions which may be conceived capable of causing a rapid increase in the amount of urate of soda in the blood and so of leading to its deposition in the joints and of provoking an attack of gout. These are (1) the ingestion at a particular time of such food as tends to "disorder the liver" and to produce lithæmia, and (2) the failure of the kidneys to maintain an adequate excretion of uric acid. The first of these conditions is well known to be a frequent exciting cause of a gouty seizure. Thus, Sir Charles Scudamore mentions the case of a gentleman without any hereditary tendency and with no reason to suspect that he would be attacked, who was seized with gout for the first time after three or four days of excessive conviviality, in which he drank freely of champagne. Many other cases are referred to by him in which persons already subject to gout were attacked within a few hours after indulgence of this kind. One striking case is that of a gentleman who had never had gout in the summer and who, persuaded of his security, drank six or seven glasses of champagne; in twelve hours he had a fit of the gout. In three instances he had heard of patients who had sat down to convivial dinners with scarcely the sensation of gout, but who when rising to leave the table found themselves completely disabled.

Dr Garrod, however, is disposed to think that in many cases the other condition (that of the kidneys) is really the one which is operative.

He found that in severe cases of acute gout the amount of uric acid contained in the urine was on an average less than four grains daily (the normal amount being eight or ten grains). It is true that in such cases the urine as it cools often deposits urates, which are of a bright pink or red colour such as has generally been supposed to be characteristic of lithæmia, and to indicate an excessive secretion of these salts. But Dr Garrod shows that this last conclusion is fallacious. The fallacy lies partly in the fact that the quantity of urine passed in the twenty-four hours is diminished, partly in the fact that its acidity is much increased, so that the whole of the urates present in it are deposited. As gout becomes more and more chronic uric acid is excreted by the kidneys in diminished amount even in the intervals between the attacks, and in advanced stages of the disease it may be entirely absent from the urine. But the *blood* in chronic gout is always rich in uric acid.

It still remains for us to inquire why one part of the body more than another should be selected for the deposition of the crystalline urate of soda. And Dr Garrod's hypothesis is that the crystals are formed especially in those parts which are not vascular or in which the circulation is sluggish. This applies both to the pinna of the ear and to the articular cartilages, and in reference to the former he says that persons with cold ears are most apt to have gouty concretions in them. In the knee-joints, too, the parts which are in contact with the vascular fringes remain free.

The reason why the metatarso-phalangeal joint of the great toe should be attacked by gout in preference to all other joints is supposed by Dr Garrod to be the fact that it is subjected to so much pressure from the weight of the body, and also to sudden shocks. I have already mentioned that when any other joint has been previously injured it is not infrequently made the victim of a gouty seizure before the great toe.

Ætiology.—In regard to the *causes* of gout, there are still some points that require notice, besides those that I have already alluded to in a former part of this volume (p. 252). In the first place, the disease is distinctly hereditary, as, indeed, is lithæmia in general. Dr Garrod finds that more than half of his gouty patients can distinctly trace an inherited disposition to this disease; and if he were to take only persons belonging to the upper classes, he says that the proportion would be considerably greater. Mr Jonathan Hutchinson ('Med. Times and Gaz.,' 1876) has observed that the younger children in a family are more likely than the elder to suffer, and to suffer severely; and there are obvious reasons why this should be the case. The popular idea that the disease often skips over a generation is believed by him to be unfounded, except for the circumstance that the child of a very gouty patient, having before his eyes the fear of suffering in the same way, may live so as to keep the disease at bay. There is a particular period of life at which a first attack of gout is especially apt to occur, namely, between thirty and thirty-five years of age. In persons less than twenty years old the disease is very rare; but Dr Garrod says that he has seen it in the great toe in youths of sixteen. In early cases there is always a strong hereditary predisposition, and the habits of the individual are also generally such as favour its development. On the other hand, beyond the age of sixty-five it seldom happens that a first seizure occurs. But Dr Garrod speaks of one case in a patient who was over eighty, and of another in a female who was in her ninetieth year. Gout is much more common in men than in women. This doubtless depends mainly upon the fact that the habits are more frequently such as tend to develop the disease. The occurrence of the catamenia during a large part of female life perhaps also assists in warding off gout, for it seldom attacks women until after the cessation of the menstrual function. Dr Garrod observes, indeed, that striking exceptions are sometimes met with, in which very severe gout, attended with great crippling and deformity, occurs in comparatively young women; but he adds that most of such cases are susceptible of explanation.

Concerning the kinds of food and drink which especially tend to produce gout I have not very much to add to what I have said already in speaking of the causes of lithæmia. Dr Garrod, indeed, speaks of an excessive indulgence in *animal* food as particularly likely to cause gout, whereas the fatty and saccharine principles, when ingested in excessive quantity, are supposed to be more apt to induce the hepatic disorder which probably causes an excessive production of uric acid in the place of urea. Dr Garrod himself admits that vegetable substances may tend to induce gout, by causing dyspepsia; and, as he remarks, it is extremely difficult to determine correctly the relative importance of the numerous influences under the simultaneous operation of which every human being is placed.

On the other hand, as regards the tendencies of the various alcoholic beverages to produce gout, striking evidence can be brought forward, in the very different liability to the disease presented by the inhabitants of different countries. It is true that inheritance here comes again into play; but only so as to intensify the action of other causes. The fact that malt liquors are more apt to produce gout than ardent spirits is well shown by comparing the working men of London with those of Edinburgh and Glasgow; the former drink beer and porter and are very liable to the disease; the latter drink little but whisky, and, although they are by no means sparing of this, they scarcely ever have gout. Dr Garrod says that even the pale bitter ales may, if taken freely, be the sole cause of the complaint. The rarity of gout in many of the cities on the Continent, where distilled spirits form the chief intoxicating beverage, is another proof that they have but little tendency to produce it; and Dr Garrod does not mention brandy as more injurious in this respect than other spirituous liquors. With regard to the different kinds of wine, his observations are in general accordance with those which I have already made; but he gives a caution against sherry—however dry and pure—which he says is by no means so innocent as many people imagine. Cider, according to this writer, when sweet and partially fermented, is apt to cause gout; but rough cider is comparatively harmless. With regard to the causes which render one kind of alcoholic drink more injurious than another, he thinks that no positive conclusion can as yet be drawn. According to him, neither the acid nor the sugar which many of them contain is the baneful ingredient. He appears rather to lay stress upon the greater or less tendency of these several beverages to cause dyspepsia, and on the other hand to act as diuretics.

A fact which Sydenham long ago observed, in his description of gout, is that the first attack generally occurs in the winter season; he says towards the end of January or the beginning of February. Why this should be the case does not appear very clear; but it is well known that deposits of lithates in the urine are very apt to occur in cold weather. Moreover, in speaking of paroxysmal hæmatinuria (p. 364), I remarked that in some persons at least one of the effects of external cold appears to be an excessive disintegration of the red corpuscles of the blood. It may well be that a similar action, less powerfully exerted, may, in a person already disposed to gout, throw upon the liver and kidneys an amount of work which they are unable to accomplish. In many patients, the disease returns for two or three years in the spring only; after a time, a second attack occurs in the autumn; and at length the seizures occur quite independently of season. The influence of cold in the development of such is further shown by the effects of change of climate. Dr Garrod says that a gouty man may often escape his accustomed winter attacks by going to Malta or Egypt. In hot countries, again, this disease appears to be very rare, and sometimes even unknown. Even in the south of Europe it is much less frequent than in England; but much stress cannot be laid upon these facts, since the habits of the people of different countries are very different.

Another occasional exciting cause of gout is fatigue or anxiety of mind. Sir Charles Scudamore mentions the cases of two female patients, in each of whom a severe first attack of gout was brought on by sitting up for several nights in succession, nursing a sick relation.

A very remarkable fact in the causation of gout has still to be mentioned, which was first discovered by Dr Garrod. I refer to the influence of chronic lead-poisoning. That this is a *vera causa* has been recognised by many other observers since he pointed it out; and it is sufficiently established by everyday hospital experience in London. Dr Garrod, indeed, says that about 30 per cent. of the gouty patients in his hospital practice had been exposed to the influence of lead. Some of them were painters and plumbers, others workers in lead mills, and others "composite-doll" makers. A careful inquiry into the habits of the men failed to show that they had been less temperate in their habits than other men of the same class. The way in which lead produces gout is at present doubtful. Probably the explanation of this fact involves that of another which Dr Garrod believes he has established, and which may be regarded as its converse: that persons who are already gouty are more susceptible than others to be injuriously affected by lead. He says that, in several instances, he has found those patients to be of gouty habit, or to have already had severe attacks of gout, in whom the medicinal administration of the preparations of lead has produced colic or a blue line upon the gums with unusual rapidity.

Diagnosis of gout is in some cases very easy, in others exceedingly difficult. According to many writers, the difficulty which so frequently occurs in determining the real nature of a case in which the joints are swollen and painful arises from the occurrence of inflammation intermediate between gout and rheumatism, or from the existence of both gout and rheumatism in the same patient at the same time. If, however, the view that I have taken of gout be correct, there can be no *tertium quid* between it and rheumatism. Either the affected joint contains urate of soda, or it does not. In the former case the disease is gout, in the latter it is not so. In the post-mortem room, doubts never arise as to the gouty nature of a joint affection; but during life we cannot see into the interior of the articulation; and it is not surprising that there should sometimes be a difficulty in deciding the question. On the other hand, so far as concerns the simultaneous presence of gout and rheumatism in the same patient, I do not dispute its theoretical possibility; but seeing that the anatomical characters of a rheumatic affection of the joints are mainly negative, I do not understand how one could prove its presence in addition to that of gout. The term "rheumatic gout," except when it is used to designate the disease which is more fitly called osteo-arthritis or rheumatoid arthritis, appears to me to be merely a cloak for ignorance as to the real nature of the cases to which this term is applied. But I admit that it is sometimes impossible, and often difficult, to arrive at a correct diagnosis in the matter.

I may briefly recapitulate the characters which suggest an affirmative answer to the question whether a case is one of gout. These are (1) that the small joints should be affected rather than the large, and particularly the great toe joint; (2) that the attack should begin suddenly in the night, especially if it be a first attack; (3) that the skin over the affected joint should be tense, shining, and red; with distended veins around it; that after a day or two it should be œdematous; and ultimately that it should desquamate; (4) that the febrile disturbance should be moderate, and should bear a certain proportion to the local inflammation. The sex, age, and family history of the patient have also to be taken into account; and the state of the heart and kidneys must be investigated. All those parts which are apt to be the seat of tophi must be examined. If a single deposit of urate of soda can be found anywhere, it almost settles the question. But care must be taken not to mistake other kinds of enlargement of the finger-joints for those caused by gout. Again, it is needful to distinguish from tophi in the pinna of the ear other granules which are really of a different nature; as an instance I may mention that little nodule in the edge of the helix which Mr Darwin believes to be an indication that the pedigree of man is traceable upwards to some animal with pointed folded ears. I know of one instance in which a medical man fancied that such a nodule in his own ear was a gouty deposit. In a doubtful case, it is probably advisable to apply a blister, or to remove an ounce of blood from the arm, and to test the serum for uric acid in the way recommended by Dr Garrod. I must confess, however, that I have tried the thread experiment several times, and have never yet succeeded in detecting the crystals.

It must not be forgotten, too, that there are other diseases besides rheumatism which may be mistaken for gout. Dr Garrod mentions a case in which the great toe was swollen, tense, red, and hot; in which, in fact, the joint looked exactly as though it were affected with severe gouty inflammation; but the disease turned out to be *pyæmia.*

Prognosis.—Acute gout in the joints appears never to be directly fatal; a prognosis is required only with regard to the probability of the nature of the disease, and of its ultimate effect on the health of the patient. Formerly it was deemed rather an honourable thing to have the gout; it showed, I suppose, that not only the man himself, but perhaps his father and grandfather before him, had been able to afford good living and plenty of it. Sydenham himself, after suffering from the disease for thirty-four years, speaks of it as a comfort that gout, unlike any other disease, kills more rich men than poor, more wise men than simple. "Great kings," he says, "emperors, generals, admirals, and philosophers have all died of gout."

At the present day any consolatory reflections of this kind may be checked by the consideration that all insurance offices charge an additional premium to anyone who has had even a single attack of gout. Their actuaries know well that the disease tends to shorten life. However slight it may have been, a seizure of gout is always an admonition that the patient's habits of life are incompatible with the preservation of his health. Sir Thomas Watson says that "in not a few instances men of good sense, and masters of themselves, having been warned by one visitation of the gout, have thenceforward resolutely abstained from rich living and from wine and strong drinks of all kinds; and they have been rewarded by complete immunity from any return of the disease; or at any rate, its future assaults upon them have been few and feeble." "I am sure," he adds, "it is worth any young man's while, who has had the gout, to become a teetotaler." For the earlier the age at which a first seizure occurs, the worse is the prognosis; and particularly if there be an inherited predisposition. Dr Garrod says that he has known thirty-five years elapse between a regular attack of gout in the great toe and the patient's death, which latter event took place when he was seventy years old. He has also seen several cases in which the disease, after having recurred periodically for many years, gradually declined in intensity and duration, and at last altogether disappeared.

The repetition of attacks of gout is a serious matter, if only on account of the crippled state of limbs which it induces, preventing the patient from taking exercise, and destroying his enjoyment of life. The effects of accidents are more dangerous in gouty persons than in others.

But the main risk connected with the gouty condition is its liability to induce a disease of the kidneys. Of this disease I have given a full description under the name of "chronic interstitial nephritis," or cirrhosis of the kidney (p. 473). The organ when affected by it is called the "small, red, contracted kidney," or the "gouty" kidney. It must, however, be stated that, although the renal affection in question may commonly owe its origin to gout, it certainly is not always traceable to this cause. There is one peculiarity in the appearance presented by a section of the kidney when the disease is caused by gout, namely, that fine white streaks are often seen, running in the course of the straight tubes in the pyramids; these white streaks have been shown by Dr Garrod and others to consist of prismatic crystals of urate of soda and of amorphous masses, blocking up the tubes, and also embedded in their walls.

The signs by which this affection of the kidneys may be detected have been discussed already (p. 477). A detailed examination of the urine, both chemical and microscopical, is often necessary. But merely looking at the secretion is often enough to excite a suspicion. Sydenham long ago remarked that in cases of long-standing gout the urine, "no longer high coloured, is pale and copious, like the urine of diabetes." He did not understand the significance of this, but we now know that it points to the fact that the kidneys are diseased. And we also know that hypertrophy of the heart is an almost constant attendant upon this form of renal affection, and that apoplexy frequently results. The slightest indication of cerebral mischief must therefore be watched very carefully in such cases, and it is often right to address a warning to the patient or to his friends that he should not be left alone at night, nor while out of doors.

Treatment.—Gout is one of the few diseases for which modern medicine has found a specific remedy. When Sydenham wrote he spoke of the possibility of such a discovery, which he said would delight him above all other physicians, but he knew of no specific for gout. Cullen, after him, advocated patience and flannel alone. Towards the end of the last century a secret remedy for the disease became widely known under the name of the *eau médicinale*. This was prepared according to a formula invented by a military officer in the service of the King of France, M. Husson. Its success was unmistakable, and various attempts were made to discover what plant was used in making it. In 1814 Mr Want maintained that this was the meadow saffron or colchicum, and two years later Sir Everard Home expressed the same opinion in a paper which was admitted into the 'Philosophical Transactions.' Since then, however, there has been much discussion as to the use of colchicum in gout. Perhaps it seemed to derive a taint from its original association with a quack medicine. At any rate, medical writers have vied with one another in their endeavours to depreciate its value. Some have asserted that when it acts beneficially it does so only by producing purgation; others that even when it removes the local symptoms it leaves "the disposition to the disease much stronger in the system." This last was the opinion of Scudamore, and for an outspoken denunciation of "mischievous drugs" we have only to turn to the clinical lectures of Trousseau, who advises his hearers to cross their arms and look on, doing absolutely nothing to subdue an attack of acute gout.

Very different from this is the teaching of Sir Thomas Watson and also of Dr Garrod, and so far as my own more limited experience has gone I am certainly inclined to agree with them. They both recommend that colchicum should be given. The former writer prescribes forty minims or a drachm of the colchicum wine in a saline draught at bedtime, and half a drachm more in a warm draught the next morning, repeating the sequence if the disease continues. The latter gives about twenty minims of the wine every six hours. Both say that the effect is almost magical; the pain is calmed, and the swelling reduced often within a few hours.

It is certain that the curative action of colchicum is not dependent upon its purgative operation. In common with others I have often observed it very effectual in cases where it did not act upon the bowels at all. Sometimes, however, this remedy produces a peculiar change in the fæces, which Dr Garrod says may enable a patient accustomed to its use to tell that he is taking it although he had not known that it was in the prescription. Some have supposed that colchicum acts upon the kidneys and so cures gout, but the investigations of Dr Garrod are unfavourable to this conclusion.

I have still to consider the question whether there is any truth in the opinions that after the use of colchicum gout returns more quickly than it otherwise would, and that this medicine impairs the health of the patient. I have read Scudamore's twenty-three cases, in most of which injurious effects were ascribed to the drug. This, however, having been the *eau médicinale*, and having been taken by patients of their own accord, and without medical authority, the cases appear to me to prove very little. It seems to have been forgotten that there is a natural tendency for the attacks of gout to recur with increasing frequency, and for the patient to suffer more and more during the intervals from dyspepsia, pains in the limbs, &c. Again, it is probable, as Sir Thomas Watson observes, that the striking efficacy of colchicum in attacks of gout has often led men to disregard those precautions of regimen and diet by which alone the disease can be kept at bay in those who are subject to it. Having what they deemed a specific, they have cast aside all restraint, and before long they have had a fresh seizure.

Alkalies, again, appear to be decidedly useful in gout, and they may often be advantageously given with the colchicum. Dr Garrod has shown that the blood is less alkaline in gout than in almost any other disease. Probably the best way in which these remedies can be given is in the form of salts of citric, tartaric, or acetic acid. And, in the hope that the base may combine with uric acid and lead to its elimination by the kidneys we may perhaps wisely adopt Dr Garrod's advice, and prescribe potass rather than soda; for the urate of potass is much more soluble than that of soda, and potass also possesses diuretic properties. The salts of lithia were introduced by Dr Garrod as possessing some advantage over those of potass in the still greater solubility of the urate of that base; but I am not aware that this is of so great gain as to outweigh the disadvantage of their much greater price. The carbonate of lithia may be given in doses of five to ten grains dissolved in aerated water, or the citrate in doses of eight to twelve grains or more. Whatever alkaline salt is prescribed should be largely diluted with water, and should be taken at least an hour before meals, at times when the stomach is empty.*

The *diet* in an attack of acute gout should for the first few days consist simply of milk, arrowroot, sago, tapioca, and the like, with water or toast and water. A little brandy may be given if the previous habits of the patient render it necessary; but not otherwise. It must, however, be added that even a first seizure of gout sometimes occurs in a patient so broken down in constitution as to require as much nourishment as the stomach can readily dispose of; such as beef tea, soup, and eggs.

The *local treatment* of gouty joints should be as simple as possible. They may be covered with flannel or cotton wool; or, as Dr Garrod recommends, oil-silk or gutta-percha sheeting may be so applied as to keep in the moisture exhaled from the skin, and form a kind of vapour bath. Some care, he says, is necessary in adjusting the oil-silk so as to completely close it; for otherwise the part becomes heated, and the pain increased. A solution of atropine and of hydrochlorate of morphia may be used, as a sedative, in the proportion of one grain of the former, and eight grains of the latter to the fluid ounce; this liquid being applied on a small piece of lint beneath the oil-silk. I have found a lotion containing a drachm of the spiritus etheris sulphurici to six ounces of water serviceable in some cases in which I have used it. Sir Charles Scudamore recommended a lotion composed of one part of alcohol and three of camphor water; this he directed to be made "just agreeably lukewarm by the addition of a little boiling water," and to be applied on thick linen compresses, which were to be frequently changed.

Dr Garrod gives a strong caution against the application of leeches to joints affected wth gout. He has seen several instances in which irremediable injury resulted from their use. Thus he speaks of great toe joints which have become stiffened after a few attacks, when local depletion had been resorted to, and of two cases in which the patients completely lost the use of both knee-joints under similar circumstances. Hot poultices, again, are said by Sir Charles Scudamore to be very injurious; he says that they increase the œdematous swelling of the affected part, and cause subsequent debility.

Another plan of treatment which was allowed by no less an authority than Harvey himself, but which appears certainly to be mischievous, is the application of cold. Sir Thomas Watson mentions that Dr Parry, of Bath, had at one time two patients under his care, each of whom had attempted to cut short or to ease a paroxysm of gout by plunging the affected part into cold water. This gave instant relief to the pain, and the inflammation presently abated, but in each case hemiplegia occurred a few hours afterwards. Trousseau relates a case that occurred to Dr Demarquay of a gentleman who applied cold water compresses to his foot, which was affected with very severe gout. The pain was almost immediately relieved. A few hours later the patient fell into a state of apoplectic semi-stupor. The cerebral symptoms disappeared under the use of sinapisms to the foot, which restored the articular inflammation. These cases certainly resemble one another so closely that it is difficult to believe that the occurrence of the cerebral disturbance in them was a mere accident. They might very fairly be cited as examples of what Cullen described as a distinct form, *retrograde* or *retrocedent* gout.

Of this variety of the disease I have hitherto made no mention, believing that, like "suppressed" gout, it would generally be more simply regarded as one of the many phases of "lithæmia." I certainly think that this is so with the affection which was formerly called "gout in the stomach."

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* [Sarcosin, which diminishes the excretion of uric acid, has been recommended on theorical grounds as a remedy for gout by Schultzen.—ED.]

The *treatment of chronic gout* must be in some respects different from that of an acute attack. But it is a great mistake to suppose that colchicum is useless, even when the joints are deformed and crippled in an extreme degree. I remember a case of this kind, in a man who lay in bed week after week until I prescribed this medicine, which I had at first supposed to be little likely to be of service to him; in a very short time he was free from pain, and about the ward.

Other remedies which are very useful in certain cases of chronic gout are guaiacum and iodide of potassium. Dr Garrod says that he has given the first of these medicines extensively, especially for the asthenic gout of old subjects; a patient may go on taking it for a whole year at a time. The second is particularly useful when the pains are increased at night, and by the heat of the bed; and also when the joints contain fluid of which the absorption is slow. Cinchona bark and quinine are also useful in certain cases.

Certain *baths and mineral springs* have so great a reputation for the relief or cure of gout that perhaps there is no patient, among the richer classes, who does not sooner or later visit some one of them. And as they may be very beneficial in some cases, and very injurious in others, one ought to know the main indications for their use. Of such resorts the chief is Vichy; the main ingredient of its springs is carbonate of soda in the proportion of about forty grains to the pint. Another very similar water is that of Vals. Now, persons who are robust and of full habit often derive much benefit from these springs; but to feeble patients they often do harm. Dr Garrod says that in very chronic cases, when there is a tendency to the rapid formation of concretions about the joints, this tendency has appeared in his experience to be rather favoured by the use of Vichy water. Trousseau says that as a general rule alkaline waters should not be taken for more than ten or twelve days consecutively, and only in small quantities. No year passed in which he did not see evil consequences from the use of such waters.

Other rules with regard to the treatment of gout by mineral waters are that they should never be taken when an acute attack is present or threatening; nor by patients who are the subject of organic disease of the heart or kidneys.

In those who exhibit symptoms of hepatic disorder, the waters of Carlsbad and the other springs mentioned under lithiasis (p. 256) are often very useful. On the other hand, when the circulation is sluggish or the secretions deficient, Wiesbaden is indicated, the water of which contains a large amount of chloride of sodium; or Aix la Chapelle. And old or infirm persons may be sent to Bath or Buxton for baths, which are of an elevated temperature and of which the active saline ingredients are comparatively small in quantity; or Teplitz or Gastein may be chosen, if these places be not too far distant.

ACUTE RHEUMATISM

Name and definition—Onset, course, and symptoms—Anatomy of the inflamed joints—The pyrexia, sweating, &c.—Cardiac complications—Pathology of rheumatism—Treatment by older methods—Observation of the natural course of the disease—Treatment by salicin and salicylates—Hyperpyrexia in rheumatism, and its treatment—Diagnosis, particularly from pyæmia.

Definition.—By ancient writers, from Hippocrates downwards, the words *rheuma* and *catarrh* are used as having similar meanings, their etymology being also alike, since the one term was derived from ῥέω (I flow), while the other came from the same verb with the prefix κατὰ. The notion was that of an acrid humour, generated in the brain and distributed over the body. In the course of time, diseases of the mucous membranes became known as catarrhs, while the name of rheumatism was applied to painful affections of the limbs and elsewhere, on account of their being so often of an erratic character. Baillon or Ballonius, who lived about two hundred and fifty years ago, is said to have taken a principal part in giving it this meaning, and he also distinguished it from gout, or (as it was then termed) arthritis. Gradually the conception of rheumation became modified so as to include a factor widely removed from the original idea of it, namely, its production by external cold. All sorts of complaints have accordingly been termed rheumatic; some, of which the cause was unknown, because they were painful; others, although painless, because cold was the supposed cause. It is, however, essential that the term should cease to be employed with these vague meanings; and I shall endeavour to use it for a single disease only. Of this, in accordance with the usual practice at the present day, I shall speak as "acute rheumatism." But I shall nevertheless include under the same name all such cases as are commonly spoken of as "subacute," for they appear to me to have no claim whatever to a separate designation.

With regard to the so-called "chronic rheumatism" there is more difficulty. I shall of course describe separately the complaint which is known as *arthritis deformans*, and which probably bears no relation whatever to acute rheumatism. But there are other cases in which a chronic joint affection has clearly arisen from, and can only be described as, a sequela of the acute disease; and it is impossible to deny that a similar affection may not sometimes be chronic from its very commencement, and yet be of the same nature. The use of the adjective "rheumatic" ought, of course, to be limited in precisely the same manner as that of the substantive. But in practice it is, I think, difficult to avoid employing it more loosely for various affections of the muscles and of other parts, of which the only common character is that they are caused by cold. Thus it is hardly possible not to speak of "rheumatic myalgia," even if one does not say "muscular rheumatism;" and authors describe a rheumatic iritis and rheumatic diseases of the nerve-trunks, &c. One cannot insist too strongly on the fact that when so used the term implies nothing as to the existence of any connection between them and true "acute rheumatism."

Acute rheumatism is often termed "rheumatic fever" in this country; the name is not a bad one, and is quite free from ambiguity. In Germany several cumbrous appellations have of late been framed for it, such as "Rheumarthritis," "Polyarthritis synovialis" (Hüter), and "Polyarthritis rheumatica acuta" (Senator).

Onset and course.—The beginning of the disease is commonly rather gradual. For a day or two the patient feels uncomfortable and perhaps complains of shooting pains in the limbs. There is seldom well-marked headache. Pyrexia gradually sets in, sometimes with a rigor, sometimes with a succession of slight chills. Presently one of the joints, and generally a large joint, is found to be swollen as well as painful. But there are other cases in which the joint-affection is the first thing which is noticed.

Some years ago certain English physicians—Dr Chambers, of St George's Hospital, and Dr Francis Hawkins—endeavoured to draw a distinction between two varieties of acute rheumatism, one of which they termed "fibrous," the other "synovial." Their views will be found in detail in Sir Thomas Watson's 'Lectures.' They were, however, altogether mistaken in supposing that the disease ever attacks the parts outside a joint rather than the articular cavity itself. It is not, indeed, easy to determine clinically the fact that fluid is effused into the interior of the shoulder-joint, the elbow, or the ankle. There may be a more or less elastic swelling at those points where the synovial membrane is most able to yield, but one may hesitate to assert absolutely that it cannot be due to exudation into the sheaths of tendons, or into other structures surrounding the articulation. But in the knee very small quantities of fluid may be detected with certainty. One has only to grasp it gently between the two hands, pressing the sides of the synovial cavity upwards, so that the fluid may accumulate beneath the patella; a slight tap upon this bone with one forefinger will then bring it down upon the condyles, giving a sensation which is unmistakable. And in those exceptional cases which prove fatal, the joints which have been affected are found at the autopsy to show distinct signs of inflammation. The synovial membrane is not, indeed, always injected, but there can be no doubt that in this, as in so many other tissues, redness may subside after death. Senator even says that fluid exudation sometimes disappears, or becomes reduced to an insignificant amount, although during life it had distended the articular cavity. However this may be, it is certain that a considerable quantity of fluid is almost always still present. This is often cloudy, with floating shreds of fibrin. Not infrequently a separable layer of lymph lines the synovial membrane, which is then very deeply reddened; or it may cover the surface of the cartilages, as, for example, in the knee-joint. Leucocytes seem to be always discoverable in the exudation, but I do not find any instance recorded at Guy's Hospital in which it has assumed the appearance of pus. In one case, which was examined by Dr Moxon, the sheaths of the extensor tendons of the wrist were full of opaque serum and of masses of greenish-yellow lymph. Articular cartilages have sometimes been found eroded, but in all probability this was accidental and unconnected with the rheumatism.

That the anatomical changes in the joints in this disease must be slight in comparison with those which occur under other circumstances might, indeed, be inferred from its clinical history. One of its most striking features is the rapidity, and even suddenness, with which it flies from one part to another. Thus a joint which is extremely swollen one day may be quite normal on the next day; and a little later it may be again affected as severely as before. There is not always any obvious reddening of the skin; generally some ill-defined pink patches or striæ are seen, but they are often near the articulation rather than over it. Sometimes, however, there is a broad, shining, crimson surface, which may pit upon pressure, and then is hardly distinguishable in appearance from that caused by gout, unless it be by the fact that the surrounding veins are not dilated. Tenderness is often very great, so that the patient dreads the gentlest touch. He may lie perfectly helpless, unable to feed himself, or even to turn in bed; if the joints of the lower jaw are affected he can scarcely open his mouth. Hospital patients commonly speak of having lost the use of their limbs, or of being paralysed. The pain is usually worse by night than during the day. Dr Garrod, in 'Reynolds' System,' remarks that the different joints are often attacked in symmetrical order: the right ankle, then the left; the right knee, then the left; and so on. But in many instances the distribution of the disease is altogether irregular. Sometimes several articulations suffer in one limb, and none elsewhere. The knuckles and other small joints are often affected, but perhaps never without some one of the large joints being also affected. According to Lebert acute rheumatism sometimes attacks the synchondroses, as, for instance, those of the pelvis.

In some exceptional cases rheumatic inflammation of a joint, instead of subsiding, passes on into a chronic stage, and ends either in ankylosis or in the disorganisation of the articular structures. A patient, for example, may be admitted into a medical ward for an acute attack, involving a large number of his joints. As he gets better, it may be found that some one of them is becoming the seat of permanent mischief, so that it may be necessary to transfer him to the care of a surgeon. I think I have seen the wrist affected in this way more often than any other articulation.

The *pyrexia* of acute rheumatism is in the majority of cases severe in proportion to the number of joints involved and to the intensity of the inflammation in them. But to this rule there are many exceptions. Sometimes the articular affection is well marked while the temperature is scarcely raised at all. In other cases there is considerable fever, whereas very few of the joints suffer, and these but slightly. Indeed, as Graves long ago pointed out, it is possible for the fever to occur alone, without any joint-affection at all. The case which he cites was one of relapse, the patient having previously passed through two attacks of the usual kind; but I believe that the converse may occur, and that an attack of apparently simple pyrexia may be followed by a relapse which is attended with joint-affection, and thus reveals its real nature. It is therefore certain that acute rheumatism is a *general* disease with localisation in the joints, rather than a local disease of the joints with symptomatic fever. Yet even Wunderlich was unable to recognise any typical course; the maximum temperature, which is usually about 104° F., sometimes occurs as early as the fourth or even the second day, but more often not until the end of the first, or the beginning of the second week. A point on which Senator lays stress is that the *pulse* is often rather disproportionately rapid, this being doubtless due to nervous excitement from pain; it may also be of a full and bounding character. But in some cases the pulse is but little accelerated. The *tongue* is generally flabby, marked by the teeth at its sides, and coated with a thick white fur. There may be thirst and anorexia, but Dr Gowers remarks that the appetite is frequently good, the patient having no nausea, and no disgust at food. So also he may be able to sleep, if not kept awake by the pain. He is seldom delirious, unless his cerebral organisation is naturally very sensitive, or unless he has been a drunkard, in which case a kind of delirium tremens is often developed as a complication. There is no tendency for the fever to assume a "typhoid" character. The *urine* is scanty, dense, high coloured, acid, depositing pink urates in abundance, and often crystals of uric acid; sometimes it contains a little albumen for a day or two.

One of the most striking symptoms of acute rheumatism, but one which is not invariably present, is *sweating*. The patient lies bathed in water, which may saturate the sheets and may even make the blankets damp. This must, of course, carry away much heat from the surface of the body, but it cannot be said to have any obvious influence in lowering the pyrexia or in relieving the articular pains. The perspiration has usually a very sour smell, which is often taken into account in diagnosis. This, however, seems to be due to the presence of some acrid material, rather than to there being an unusual quantity of free acid in the fluid. For Dr Garrod says that in several cases he found the perspiration less acid than in healthy subjects; and Sir William Gull used to point out in his wards at Guy's Hospital that the reaction to litmus paper often varies upon different parts of the skin, being acid, alkaline, and neutral in the same patient. I do not know whether these differences can always be accounted for by supposing that the sweat has become alkaline by conversion of urea into carbonate of ammonia. Senator says that this decomposition often occurs between the toes and in the armpits. There is frequently developed a copious eruption of *sudamina*—minute transparent vesicles, containing an acid fluid which is no doubt sweat. They may glisten when a bright light falls upon them, but they are colourless, so that they often can more easily be felt than seen. In many cases, however, their bases become slightly inflamed and reddened, and their contents opalescent and alkaline. Writers then, without any necessity, change the name of the cutaneous affection and speak of it as *miliaria*. That it bears no special relation to acute rheumatism is shown by the fact that a precisely similar eruption occurs in other diseases attended with sweating, and also in healthy people during very hot weather. I have known a woman come to the hospital and obtain admission into the clinical ward for this complaint alone.

In this connection it is perhaps worth while to mention the "febris miliaris" which occurred as an epidemic on the Continent, and especially in France, throughout the eighteenth century and down to the year 1856; it was attended with a red papular or vesicular rash. In the English "sweating sickness," of which there were five terrible epidemics between 1485 and 1551 in this country, there seems to have been no such eruption;* at least, Dr Guy makes no allusion to it in the graphic description of that disease in his work on public health.

In some cases of acute rheumatism there is another very remarkable eruption, the characters of which vary rather widely, but which generally assumes the form of an erythema, an urticaria, or a purpura. It has often been described under the name of *Peliosis rheumatica,* originally invented by Schönlein. Dermatologists have broken it up into several varieties, which they have termed "Erythema papulatum," "E. tuberculatum," "E. circinatum," "E. marginatum," &c., according to the appearance it happens to present; and another modification of it, as I believe, has been set aside by them under the designation of Purpura urticans. It appears to me that the best name is that proposed by Hebra, "*Erythema multiforme;*" and I adopted it in cataloguing the models of diseases of the skin in the museum of Guy's Hospital, where there are several striking examples of the affection. I must mention, however, that Scheby-Buch, of Hamburg, has recently maintained (in the 'Deutsch. Archiv' for 1874) that the relation to acute rheumatism is only apparent; the joint affection he declares to be really analogous to that which occurs in scorbutus or in hæmophilia. But in two of the cases from which our models were taken there was pericarditis; one of the patients had had a previous attack of acute rheumatism, and the other was admitted for chorea. I have myself repeatedly seen an eruption of *erythema multiforme* developed in the course of an ordinary attack of acute rheumatism, the nature of which seemed to be unequivocal. But sometimes, no doubt, it appears as a substantive disease, the joints being either unaffected or but slightly swollen and painful. That in all such cases its nature is the same I will not assert, although I think that probably this is the case. The eruption itself consists of sharply-defined spots and patches, of a purple colour rather than bright red, sometimes forming rings or presenting circinated borders. A few wheals are often scattered among them, or the appearance may be altogether that of *urticaria.* The patches generally become ecchymotic, and pass into yellow and greenish tints before they disappear. But sometimes there is a regular purpura, even the wheals appearing as purple-black tumours. The backs of the hands and of the feet are favourite seats of this affection, as was long ago pointed out by Hebra; but it may also spread over the forearms and the legs, the arms and the thighs, and occasionally the trunk and the face.

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* [See the account given by Dr Kaye (the founder of Caius College) in his tractate "De Ephemera Britannica." The years were 1485, 1506, 1517 (the worst, according to Stowe) 1528, annd 1551.—Ed.]

Cardiac complications.—In every case of acute rheumatism it is necessary, from day to day, to watch with the stethoscope for indications of inflammation of the pericardium or of the valves of the heart. These affections have been already described; but I must here insist on the fact that they often give rise neither to pain, nor to dyspnœa, nor to any other subjective symptom which might draw attention to them. And, on the other hand, Senator remarks that attacks of palpitation and faintness and oppression at the chest sometimes arise and last for some hours without any cardiac lesion being present. The frequency of inflammation of the heart in acute rheumatism cannot, I think, be stated with numerical accuracy. The results of post-mortem examinations are not available for the purpose, because cases which prove fatal are more severe than the average; and, on the other hand, the auscultatory signs which afford our best means of detecting cardiac complications during life are open to certain sources of fallacy. Probably, however, we shall not be far from the mark in estimating that from 50 to 60 per cent. of all cases are accompanied by pericarditis or by endocarditis, or by both. Among forty-five cases which ended fatally at Guy's Hospital, and in which there had been no previous or chronic disease of the valves, I find that the heart was healthy in eight only, that both pericarditis and endocarditis existed in nineteen, pericarditis alone in ten, endocarditis alone in eight. These figures differ widely from those which Dr Sibson gave as the result of an analysis of 326 unselected clinical cases in his elaborate article in the fourth volume of 'Reynolds' System.' According to these, endocarditis was supposed to have been present fully three times as often as pericarditis.

There is no doubt of the fact that acute inflammation of the heart is sometimes, and especially in children, the sole indication of a rheumatic attack, and that in other cases it is attended with but very slight and fugitive joint-pains, which may easily escape notice altogether. Nevertheless, as a rule, there is in acute rheumatism a direct relation between the degree of severity of the articular affection and the frequency, as well as the intensity, of cardiac complications. Thus Dr Sibson found that the joints were severely attacked in only one fourth of those cases in which the heart showed no sign of inflammation, but in two fifths of those in which simple endocarditis was believed to be present, and in three fifths of those in which there was pericarditis, with or without endocarditis.

Age, however, has a very marked influence upon the frequency of inflammation of the heart in acute rheumatism. The younger the patient, the greater the liability to it; in young female servants, who make up a large proportion of the cases seen in hospital practice, the heart scarcely ever, according to Dr Sibson, fails to show some signs of being attacked.

On the other hand, many older patients may escape; and here a curious difference is found to exist between males and females. Pericarditis seems to be three times as frequent in men above the age of twenty-five as in women of corresponding age. The reason for this is believed to be that a laborious occupation greatly augments the liability to cardiac complications in acute rheumatism; most women who are not young work less hard than men of the same age, whereas the girls of the lower classes are very apt to have their strength overtaxed.

Of the other complications of acute rheumatism, *pleurisy*, affecting chiefly the left pleura, is the most frequent. Lebert speaks of it as occurring in 10 per cent. of all cases; and it would probably be far more often diagnosed than it is, if the pain produced by moving rheumatic patients did not interfere with stethoscopic examination of the back. There is sometimes bronchitis. The lungs are often œdematous, and, in fatal cases, the microscope may show them to be affected with slight catarrhal pneumonia. On the other hand, anything like hepatisation, of even a part of one lung, seems to be of extremely rare occurrence. I can hardly find a single instance in which it was found to be present on post-mortem examination.

In one fatal case that occurred at Guy's Hospital the *tonsils* were suppurating, and acute tonsillitis sometimes begins an attack.

Ætiology.—In causing acute rheumatism a very important factor is *cold.* The patient is not infrequently attacked within a few hours after getting wet through; and even when there has been a longer interval, of perhaps a week or a fortnight, since such an occurrence, I do not myself doubt its having been really the cause of the disease. But sometimes the exposure may have been repeated again and again for a considerable time before any harm seems to result. I am not sure whether in such cases the cold should be supposed to have a cumulative action, exerted slowly and from day to day; or whether the reason of the delay is not rather the varying state of the patient, who does not suffer until his power of resistance is so diminished by over-fatigue, or in some other way, that he now for the first time gets "a chill." I recently had a general servant under my care, who during the whole of one December had been obliged to sleep in a very damp washhouse because her mistress had chosen to take in a lodger; she was attacked at the end of about a month with acute rheumatism. Some years ago a footman was admitted into hospital from a house in Eaton Square; he attributed his illness to sleeping in a damp room, and he said that more than one of those who had previously held the same position had suffered in a similar way. Some other conditions which affect the frequency of the disease are probably operative only in so far as they increase or diminish the chances of exposure to cold. Thus, acute rheumatism is less common during the months of July, August, and September than at other seasons of the year.* It is true that the number of patients admitted into the wards of hospitals in this city at different times is liable to wide variations which are not readily attributed to changes in the weather; for a few weeks hardly a case may present itself, whereas afterwards a great many may appear within a few days of one another. But it seems less reasonable to attribute such variations to an unknown epidemic agency (which could hardly be anything but a miasm) than to the greater or less incidence of cold upon the population in such a form as to be effective in producing chills.

The geographical distribution of the disease seems as yet to be too imperfectly known to enable one to state positively what are its bearings upon this question. Hirsch gives a large amount of information; but with regard to many of the statements which he cites it is difficult to tell whether acute rheumatism is meant, to the exclusion of the vague affections which are so often grouped together as "rheumatic." Thus, after stating that "rheumatism" is prevalent among the Esquimaux, in Iceland, and in Polar regions generally, he sums up by declaring that *acute rheumatism* is a disease mainly of the temperate zones. One striking circumstance which he mentions is that in Cornwall and in South Devon it is comparatively very infrequent. In dispensary practice during four and a half years in Cornwall only four cases occurred; and I remember to have been told by medical men practising in Exeter that it seldom is seen in that city. The Isle of Wight and Guernsey are said to enjoy a similar degree of immunity from it. On the other hand, in the United States "rheumatism" appears to be far more frequent in the Southern States than in the North-Eastern States, where the climate is damp and cold and changeable. The whole question evidently demands further investigation.

Among the circumstances which dispose one person rather than another to be attacked by acute rheumatism, some mean only a greater liability to be affected by cold; but there are others of which this cannot be said. Thus there can be no doubt that the members of certain families are especially liable to it; one finds in the course of years several children of the same parents are often affected by it, and that besides those who have acute rheumatism others have chorea. Dr Garrod says that he traced a *hereditary predisposition* in about a quarter of his patients. As to whether the disease is more apt to occur in persons of one complexion than in those of another, I am not so sure. According to Dr Laycock, individuals of "the rheumatic diathesis purely" are usually well built and well nourished, the complexion is of a healthy florid tint, the teeth are regular, sound, and firm, the hair is abundant; the skin is sometimes greasy and thick. Mr Hutchinson (following Bazin) is convinced that there is a diathesis which is termed by him arthritic, and which is common to gout, rheumatism, and arthritis deformans; but I do not know that he has anywhere associated it with a definite physiognomy.

A very striking feature of acute rheumatism is its tendency to recur. The interval is commonly from three to five years, but it may be far longer. Probably, however, it is not that having once had the disease disposes to its recurrence, but rather that the personal susceptibility continues which enabled cold to give rise to it on the first occasion. Accordingly, a large proportion of first attacks take place in children and in young adults; indeed, it scarcely ever happens that a person who has not previously had the disease falls ill with it after the age of fifty. In infants, however, it is very uncommon; only a few cases have been recorded, one at the the age of twenty-three days by Widerhofer, another at four weeks by Häger, and one at ten months by Henoch; references to these cases are given by Senator in Ziemssen's 'Handbuch.' It does not seem that there is any decided difference in the liability of males as compared with females to acute rheumatism.

Muscular *exertion* is another factor which is believed by many observers to play a very important part in the ætiology of the disease. It not infrequently sets in immediately after a long march, and its great frequency in domestic servants is supposed to be due to their being so often sadly overworked. Dr Sibson has even thrown out the suggestion that the joints most apt to be affected are those which take the greatest share in the habitual labour of the patient; thus he had under his care a coachman who was first attacked in the right thumb. Some of the occupations which furnish large contingents of cases of acute rheumatism are those which involve arduous muscular work; thus it is frequent in smiths and in carpenters.

Lastly, acute rheumatism is not infrequently associated with *scarlet fever*, apparently without being traceable to either cold or to fatigue. It usually appears during the stage of desquamation.* A similar affection sometimes occurs as a sequela of dysentery (p. 185). The puerperal state is also believed to bear a causal relation to acute rheumatism, which is not infrequently developed in women who have been recently confined, or have miscarried, especially when there has been profuse hæmorrhage.

Theory of rheumatism.—The relation which appears to exist between rheumatism and excessive muscular exertion is of great interest, because it may possibly throw light upon the pathology of the disease, which as yet is exceedingly obscure. Some writers have imagined that it may in some way depend upon disturbance of nervous centres; others that it may be of embolic origin. Neither of these hypotheses seems to me to merit serious discussion. The former is in the highest degree vague and supplies not a single link to bind together the phenomena of acute rheumatism; the latter is at variance with all that is known concerning the real effects of embolism, and with the fact that endocarditis is often absent, and when present is secondary to the joint affection. On the other hand, I have always been attracted towards a third view, which was originally suggested by Prout, and according to which an acid, probably lactic acid, is supposed to be concerned in the disease. It is true that the peculiar sour smell of the perspiration affords little or no presumption in favour of such a theory, now that we know the reaction to test paper. Nor perhaps is there much force in the fact that gout, which resembles acute rheumatism in some points, is due to the deposition of uric acid in combination with soda, in the articular cartilages. In 1858 Dr B. W. Richardson published a series of experiments upon dogs and cats, which were believed by him to show that the injection of lactic acid into the peritoneal cavity was capable of setting up endocarditis; but three years later Reyher, in 'Virchow's Archiv,' pointed out that appearances precisely similar are constantly seen in the cardiac valves of dogs on which no such operation has been performed. After this the lactic acid theory languished, until in 1871 Dr Balthazar Foster, of Birmingham, recorded in the 'British Medical Journal,' two cases, in each of which the administration of the acid (in doses of ♏xv to ♏lxxv) with a view to check diabetes, was followed by the occurrence of painful swellings of the joints; one patient had no fewer than six well-marked attacks, the symptoms of which seem to have borne the closest possible resemblance to those of acute rheumatism. Külz in his 'Beiträge' has since related a case in which lactic acid set up pains called rheumatic, in the left hip and thigh. It is true that the same substance has been repeatedly administered to other patients and to healthy persons without any such result. But, as Senator remarks, such negative observations signify little or nothing, since no one can doubt that a "personal susceptibility" to the disease is an essential part of its ætiology. The most plausible conception seems to be that under the influence of cold the lactic acid which is always formed as the result of muscular exertion fails to be destroyed by oxidation or perhaps to be excreted by the skin, as it normally should be, and that it accumulates and acts as an irritant to the tissues of the joints. Senator cites, as having perhaps a bearing upon the development of acute rheumatism after muscular exertion, the observations of Frerichs (recorded in Wagner's 'Handwörterbuch der Physiologie'), according to which the synovia has a different composition in cattle that have been allowed to graze in the fields, from that which it has in cattle that have been fattened indoors, containing much more albumen and mucin in the former.

Treatment.—The natural tendency of acute rheumatism is, in the immense majority of cases, to subside after a longer or shorter time, so that the patient gradually regains his former health, except for the damage which has too often been done to the heart. And, in reference to treatment, a careful study of the course of the disease is of extreme importance, since, as I believe, it now furnishes one of the most striking instances that can be given of therapeutical success, established on a solid statistical basis. Until lately, however, nothing could be more unsatisfactory than the treatment of acute rheumatism. A great variety of medicines were prescribed, each of which appeared to be highly successful in some cases, while it failed very conspicuously in others. Some physicians, and especially the late Dr Fuller, of St George's Hospital, recommended the administration of alkalies, in such doses as to maintain an alkaline reaction of the urine ; some, following my colleague Dr Rees, pinned their faith to lemon juice. Some gave quinine, others the tincture of iron, others colchicum, others ergot, others propylamine [or rather triethylamine]. Dr Herbert Davies advocated the employment of blisters to all the affected joints; surrounding the knees, for example, with strips of emplastrum cantharidis three inches wide. It seems to be certain that the blistering plan is often followed by a rapid subsidence of the local inflammation, but there is the disadvantage that when carried out fully it is apt to produce strangury, and (as Senator has shown in vol. lx of 'Virchow's Archiv') sometimes leads to the presence of fibrin in the urine, which may not only contain flakes when voided, but even coagulate afterwards. And it may produce local sloughing.

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* [See papers by Dr Thos. Barlow and Dr Ashby, 'Brit. Med. Journ.,' Sept. 15, 1883.—Ed.]

It would be difficult to say when observers of disease first began to entertain the suspicion that the apparent success of various methods of curing acute rheumatism might, after all, be fallacious, and that the result might not have been different, even if nothing had been done. Sir Thomas Watson used to cite, but without assenting to it, the dictum of the first Dr Warren, who, when asked what was good for acute rheumatism, replied, "Six weeks." But it is only within the last few years that careful observations have been systematically made and recorded of cases left to themselves, or in which, at any rate, no drugs have been administered that could be supposed to disturb their progress. Such an "expectant treatment," as it has been absurdly termed, seems to have been first tried by Lebert, who in 1860 published the results which he had attained in nine cases. A few years later Sir William Gull and Dr Sutton, in the 'Guy's Hospital Reports' for 1866, placed on record the details of twenty-five cases.

From these observations it is clearly apparent that acute rheumatism is altogether unlike those among the specific fevers which have a fixed or definite duration. Among Sir William Gull's patients the length of time during which active symptoms continued, including that passed before admission as well as subsequently, varied from nine to thirty-four days. And, on striking an average, the course of the disease, as measured by the period at which there is freedom both from pyrexia and from joint-pains, turns out to be much shorter than that named by Dr Warren. The English physicians made it 19 days ; Lebert gave 16 days as the average time when the symptoms first greatly improved, 22 days as that for convalescence.

In attempting to compare cases of acute rheumatism left to themselves with those submitted to treatment by drugs there is a fundamental difficulty. Strictly speaking, the treatment ought to be begun on the very day on which the patient is taken ill. But to exclude all other cases would be not only to limit the field of observation almost entirely to private practice, but also to ensure that the cases accepted should be of far more than average severity, since only patients seized with violent symptoms are likely to seek medical advice at once. It is therefore impossible to reject cases which have come under notice at varying periods. But now arises the question whether one should take into account the time which the disease has lasted before the patient is seen. It is clear that if treatment is effective the fact must be brought out most strikingly when this time before treatment is left out of consideration; on the other hand, if drugs are useless, we should take the whole duration of the cases as a basis for comparison.

It is, indeed, easy to prepare both sets of figures, as was done by Gull and Sutton. And, in dealing with average results, no serious error is likely to arise whichever set be adopted, provided the cases are numerous, so that each group may contain its proportion of patients brought under treatment at an early stage, and of those in whom the disease was already far advanced.

But it seems to me that there is an insurmountable objection to the employment of averages, which not only must invalidate the comparisons made by Gull and Sutton of the results obtained by the "expectant" with those by other methods, but also render all such figures useless as standards for reference in future. The objection is that some cases of acute rheumatism run a very protracted course, which, instead of terminating in a few weeks, is measured by months. One cannot be surprised that no such instances should be found in Gull's and Sutton's papers; for it would be scarcely practicable to keep the patients week after week under observation without attempting something for their relief; and afterwards, in tabulating results, the very fact of treatment having been adopted would be sufficient to exclude them. But when one is dealing with a series of cases submitted to any particular treatment, it becomes impossible to reject the cases in question, some of which may even have been discharged from hospital still uncured. And nothing is more likely than that when an average is taken they should swamp the other cases in which treatment may have been successful.

I think, therefore, that the only fair way of using Gull's and Sutton's cases is to tabulate the length of time that symptoms lasted after admission: the list is then as follows, a fatal case being excluded.

TABLE I.—*Natural Course.*

Duration of symptoms while in hospital.	Number of cases.	Duration of symptoms before admission.	
		Figures in the several cases.	Average.
1 day	0	—	—
2 days	0	—	—
3 days	1	21 days	21 days
4 days	1	5 days	5 days
5 days	1	Indeterminate	—
6 days	2	7 days, 5 days	6 days
7 days	3	3 days, 3 days, 14 days	6·6 days
8 days	1	5 days	5 days
9 days	1	9 days	9 days
10 days	2	8 days, 5 days	6·6 days
11 days	3	8 days, 5 days, 5 days	6 days
12 days	2	6 days, 10 days	8 days
13 days	1	6 days	6 days
14 days	1	14 days	14 days
15 days	0	—	—
16 days	2	12 days, 6 days	9 days
17 days	0	—	—
18 days	1	12 days	12 days
19 days	0	—	—
20 days	0	—	—
21 days	1	4 days	4 days
27 days	1	7 days	7 days
Totals .	24		
Duration before admission of 23 cases in which it could be determined		180 days	
		Average in the 23 cases . .	7·8 days

It is, of course, to be wished that the number of cases had been larger; but from the regularity with which they are distributed over different parts of the column one may perhaps conclude that the result is fairly representative. A point which comes out clearly from the last column is that a comparatively long duration of the disease after admission is by no means peculiar to patients admitted at a very early period of their illness. On the other hand, it may be noticed that the case which subsided most rapidly while in the hospital had already been running on for three weeks. And I am not sure that this was a mere accident, for on tabulating ten other cases which have since occurred at Guy's, and in which rapid recovery has taken place without treatment (or under treatment, which I believe to be ineffectual), I find that the average duration of the disease before admission was 13·7 days.

By way of comparison I may append the following table, which shows the results of three different methods of treating acute rheumatism, as recorded by physicians who have severally advocated them.

TABLE II.—*Results of treatment by lemon-juice, alkalies, and blisters.*

Duration of symptoms after commencement of treatment.	Lemon-juice. Dr Owen Rees ('Guy's Hosp. Rep.,' xii).	Bicarbonate of potass. Dr Garrod ('Med.-Chir. Trans.,' xxxviii).	Free blistering. Dr Davies ('Lond. Hosp. Rep.,' i).
1 day	1		
2 days	—	1	
3 days	—	3	
4 days	1	8	
5 days	—	6	1
6 days	1	6	2
7 days	2	7	1
8 days	—	5	2
9 days	1	9	1
10 days	2	—	
11 days	—	2	
12 days	—	4	
13 days	—	—	1
14 days	1	Doubtful	5
Totals . .	9	51	13

It appears to me to be clear that, according to these figures, no very striking success can be claimed for any one of the three plans of treatment. The most favourable seems to have been the alkaline method introduced by Dr Garrod; but even under this the cases in which the symptoms subsided within five days amount to only eighteen out of fifty-one, or less than 36 per cent.

Salicin.—On the other hand, I think I can show that the duration of the disease may be greatly shortened by the administration of certain remedies which have recently been introduced, namely, salicylic acid and salicin. It was in January, 1876, that Dr Stricker, assistant in Traube's Clinic in Berlin, first prominently draw attention to salicylic acid, which had been used there for some months previously; the same medicine had, however, been previously employed at Basle by Buss. Salicin was originally advocated by Dr Maclagan, of Dundee, in March, 1876, in the 'Lancet;' he had first prescribed it in 1874. Among the Hottentots and the Boers of South Africa willow tea has, according to Mr F. Ensor, long been a traditional remedy for rheumatism.

When I first made trial of these drugs, I was for a little while sceptical as to their value. The patients rapidly recovered; but I could not forget that I had sometimes seen the administration of other medicines followed by results which appeared very striking. But when case after case recovered, with scarcely a failure, I became satisfied that I had a most potent remedy in my hands; and all further experience has strengthened me in this conviction. The immense majority of physicians and practitioners now, I think, entertain a similar opinion. I am not aware that any observer has in this country publicly expressed his dissent from it, except Dr Greenhow, to whose papers, in the Clinical Society's 'Transactions' for 1880, I shall presently refer.

I have been at the pains to tabulate not only my own experience at Guy's Hospital, but also that of all my colleagues, in the treatment of acute rheumatism with salicin or salicylic acid from the spring of 1876 to the end of 1880; and I have obtained the following results:—

TABLE III.—*Results of treatment by salicin or salicylic acid at Guy's Hospital,* 1876—1880.

Period at which patient became free from pyrexia, as well as from joint-pains, reckoned from commencement of treatment.	Number of cases.	Number of those cases in which relapses occurred.		
		1 Relapse.	2 Relapses.	More than 2 Relapses.
1 day	7	2		
2 days	41	8	1	1
3 days	40	11	4	
4 days	42	10	3	1 (6 relapses)
5 days	50	17	7	3 (in one 5 relapses)
6 days	23	4	2	
7 days	21	6	1	
8 days	20	9	2	
9 days	13	7	3	1
10 days	14	3	2	1
11 days	14	2	1	
12 days	6	2	1	
13 days	5	2		
14 days	6	0		
15 days	4	2		
16 days	2	1		
17 days	2	0		
18 days	3	1		
19 days	0			
20 days	0			
21 days	2			
22 days	1			
23 days	3	2	1	
24 days	2			
25 days	1			
26 days	2			
27 days	0			
28 days	2	1		
29 days	3	2		
30 days	1			
32 days	1			
35 days	1			
36 days	1			
37 days	1			
41 days	1			
49 days	1	1		
55 days	1			
60 days	1			
Indeterminate	15			
Fatal cases	2			
Totals . . .	355	93	28	7

These figures obviously compare very favourably with those given in Table I as the result in Gull's and Sutton's cases. The symptoms were arrested within five days in no fewer than 180 of our 355 patients, at which period only three of their twenty-four patients had lost their symptoms. It must be added that the rapidity with which relief is afforded to the joint pains is actually far greater than appears in the table. In many of the cases which are set down as having become relieved at the end of five or six or seven days the patient within two or three days was almost without pain, or had almost a normal temperature, or was almost free both from pyrexia and from pain. It is no uncommon thing for the patient to be conscious of experiencing great relief from the first two or three doses of the medicine; and house physicians have repeatedly told me of the striking contrast, even on the first night after admission, between patients treated with salicylic acid or salicin and those to whom no medicine had as yet been administered; the former lie quiet, even if they do not sleep; the latter often shriek and cry out during nearly the whole night, disturbing everyone else in the ward. Moreover, in many of the cases placed lower down in the list, the drug was given for a few days, or even for twenty-four hours only, and was then withdrawn; or the doses in which it was administered were such as are probably too small to arrest the disease. I have included every case in which as much as sixty grains of salicylic acid or of salicin was given in the twenty-four hours. With a single exception the tabulation is drawn up, in every respect, in such a way as to place the facts in the light most adverse to the success of the treatment.

Relapses.—The exception just alluded to has reference to the relapses which occurred in ninety-three of the 355 cases. I do not include in this list certain instances in which during convalescence (perhaps when the patient first got up) pain returned for a day or two in a single joint, or in which without pain the temperature rose once or twice to a point between 99° and 100°. Such occurrences were not infrequent, but as no treatment was required, and as recovery was in no way retarded, one may, I think, leave them out of consideration. The actual relapses more or less closely resembled primary attacks of acute rheumatism, so that the patients had again to be kept in bed and on low diet. In one case a relapse lasted twenty-one days; and the average duration of thirty-one relapses (some treated, some left to nature) I find to have been between five and six days. Now, there are two ways of looking at the relapses of acute rheumatism. One is to regard them as continuations of the original illness, and to suppose that it has been interrupted and postponed, but not really cut short by the administration of the remedy. This view is especially applicable to cases in which the symptoms return very soon after the discontinuance of treatment. Thus in four instances the disease reappeared within about twenty-four hours; and in nine others before a week had elapsed. Now, in a case of Dr Habershon's in which the urine was tested with perchloride of iron for several days after the administration of salicylate of soda had been left off, the purple reaction indicative of the presence of a salicyl compound was obtained as late as the eighth day; it is therefore quite possible to set down all the thirteen cases just alluded to as examples of the "recrudescence" of the primary attack. But on the other hand there were six cases in each of which the relapse began when a period of from three weeks to two months had passed after the subsidence of the primary attack, and when the medicine had long been discontinued. Such cases must, I think, be looked at in the same light as those which relapse when no treatment at all has been adopted; this happens not infrequently, but in what numerical proportion of cases it is not, I believe, possible at present to state. It does not seem to me to be likely that relapses should be more apt to occur when salicin or salicylic acid has been given, than when the disease has been treated in other ways, or left to run its own course, However, those observers who (as will presently be mentioned) consider salicylic acid a very depressing agent would perhaps be not unwilling to entertain such an opinion. The increased liability to relapse in enteric fever when antipyretic methods are adopted cannot be cited as analogous, because the two diseases are believed to differ so completely in their pathology. In twenty-eight of the 355 cases treated with salicin or salicylic acid at Guy's Hospital the original attack was followed by more than one relapse, in five of them by three, in one by four, and in one by six. It may easily be imagined that such patients spent many months in the hospital wards. I think, however, that it is quite a question whether the frequency of relapses might not be greatly diminished by systematically continuing the administration of moderate doses of salicylic acid, or perhaps rather of salicin, until after the lapse of several weeks.

Doses.—The dose of salicylic acid which is adequate to arrest acute rheumatism with rapidity appears, as a rule, to be about twenty grains, given at intervals of two or three hours. But sometimes a larger quantity is required. I recently had a patient who took twenty grains of salicylate of soda every two hours without any marked result for two or three days, but in whom the disease at once yielded when thirty grains were given. On the other hand, there are cases in which a dose of ten grains repeated every six hours seems to be perfectly effectual. It is generally necessary to use a larger quantity of salicin. Dr Maclagan recommends that from twenty to forty grains should at first be given every hour. Salicin may be prescribed as a powder, to be stirred up in cold water, and swallowed, or twenty grains may be dissolved in an ounce of warm water. Salicylic acid is now usually administered as a soda-salt, dissolved in aqua menthæ piperitæ, or in aqua carui. Or the acid may be dissolved in the solution of citrate or of acetate of ammonia (the porportion being gr. xx of acid to ʒiij of liq. amm. acet.), and sweetened with extract of liquorice.

Influence on the heart.—As regards the possible influence of salicylic acid in preventing the development of cardiac complications in acute rheumatism, I do not think that one is as yet in a position to make any definite statement. One cannot but remember that for each new method of treatment introduced within the last few years—even for Dr Davies' treatment by local blistering—a claim invariably made has been that it lessens the liability to inflammation of the heart. On the other hand, Gull and Sutton showed that in cases in which the organ is healthy at the time of the admission of the patient into hospital, it seldom becomes subsequently attacked. Still, one may reasonably expect that any remedy which possesses the power of arresting acute rheumatism, so that after its administration fresh joints no longer become affected, must also hinder the development of what is believed to be an analogous morbid process in or around the heart. And although in sixty-nine of my 355 cases auscultation revealed some changes in the character of the heart-sounds, while the patient was in the hospital, I can hardly find one in which there was reason to believe that pericarditis set in at a time when the action of the remedy was fully established. Almost all of them were cases in which at one period or another a systolic murmur was detected, such as I believe to be extremely difficult of interpretation.

On the other hand, so far as one can tell, it would certainly seem that salicylic acid has no power of controlling or arresting the cardiac complications of acute rheumatism, when they have once developed themselves.

Drawbacks.—In many cases, however, the administration of salicylates is attended with inconveniences, and sometimes with symptoms which are alarming. An effect, which is rather frequent, is nausea and *vomiting*, accompanied with pain at the epigastrium, or (if the acid has been given) with a burning sensation at the back of the throat. I have rarely found this so severe as to make a change of treatment necessary.

Another effect is *enfeeblement of the heart's action*. This, at Guy's Hospital, has, I think, rarely attracted attention; in a few instances I find it noted that the pulse became weak, and sometimes that it was irregular or intermittent; in one case it fell, after nine days, to fifty-two beats in the minute; in two the first sound of the heart became inaudible, and the heart's impulse could no longer be felt. The administration of stimulants has, however, been very seldom deemed necessary. On the other hand, Dr Greenhow says that in his patients, who were treated with salicylic acid, "more or less weakening of the pulse, requiring the free administration of brandy, occurred in nearly every case. This was accompanied by great weakening of the impulse of the heart, and in ten cases by almost complete obliteration of the first sound." In the Clinical Society's 'Transactions' for 1880 Dr Goodhart has recorded a case in which sudden death in the night took place, probably as the result of failure of the heart, for the pulse had been rising in frequency; he was rather disposed to attribute this result to the administration of salicylic acid, but I think without justice, for only sixty grains in all had been given, and none of the known effects of the drug were observed; at the autopsy early pericarditis was found.

Far more obvious than the cardiac are the *cerebral symptoms* to which salicylic acid (but not, I think, salicin) in many cases gives rise. Deafness is a very frequent and an early effect of the remedy. It is often accompanied with a sensation of giddiness, and with noises in the ears, which by the patients are described as being like buzzing or ringing, or are compared with the noise of a train or the rushing of water. In some cases there is headache which may be very intense, some patients becoming delirious, screaming and struggling to get out of bed. It is curious that in several cases a particular hallucination has been recorded, namely, that someone is under the bed on which the patient is lying. A boy who in 1878 was in Dr Pavy's ward got an idea that there was a dog under him, would not allow the bedclothes to touch him, and went into paroxysms of terror, with dilated pupils and livid lips. In a girl who was admitted in the following year delirium set in at the end of eighteen hours after the commencement of the treatment, which consisted in the administration of twenty grains of salicylate of soda every two hours. It often happens that when such effects are produced the patient has already lost his pains, and that his temperature has fallen to 99° or to 100°; the latter circumstance distinguishes the cerebral symptoms due to salicylic acid from those which may accompany the disease when no treatment is being carried out. The face may be deeply flushed and bathed in perspiration. In one case the delirium is reported to have been attended with "typhoid symptoms so that the prognosis for a time was grave." But I cannot find a single instance in which the patient did not become rational in a few hours, or in a day or two, after the medicine was discontinued; and there has not been one in which a fatal result followed. What would happen if one were to persist in the administration of the acid I do not know. It has often been resumed after a few days' interval without further ill-effects; but sometimes delirium has set in on successive occasions.

Another occasional effect, whether of salicin or of salicylic acid, is *epistaxis*. It was repeatedly noticed by Dr Greenhow, and has occurred in many cases treated at Guy's Hospital. It often recurs several times. It seems never to be attended with any evil consequences, except that it must no doubt tend to produce weakness and to retard convalescence.

Lastly, it is said of these remedies, but especially of the acid, that they cause anæmia, and that their administration usually leaves the patient exhausted and feeble, so that he regains health and strength more slowly than if the disease had been left to run its natural course. I am not sure how far those statements are well founded. There can be no doubt that the stay in hospital of cases treated with salicylic acid is little if at all shorter than it used to be before the remedy was used. But it must be remembered that for a long time after active symptoms have ceased one keeps the patient on low diet and confines him strictly to bed, for fear of the occurrence of a relapse. I am in the habit of insisting on a clear interval of a week before I allow even fish to be given, or permit the patient to sit up for an hour in the evening. I refuse to listen to complaints of hunger, or to urgent appeals for permission to dress and get about. These precautionary measures necessarily protract the case very much.

It remains for me to consider the facts brought forward by Dr Greenhow, which led him to express, on the whole, an opinion unfavourable to the treatment of acute rheumatism by salicylic acid or by salicin. On throwing his sixty cases treated by one or the other of these medicines into a form which enables them to be compared with mine, I obtain the following results:—

TABLE IV.—*Dr Greenhow's results with salicin.*

Period at which patient became free, both from pyrexia and joint pains, dating from the commencement of treatment.	Number of cases.	Number of those cases in which relapses occurred.
1 day	5	2 (in 1, two relapses; in 1, three relapses).
2 days	11	3 (in 1, two relapses).
3 days	14	6 (in 2, two relapses; in 2, four relapses).
4 days	5	3 (in 1, two relapses).
5 days	1	1 (two relapses).
6 days	1	1
7 days	1	
8 days	2	
12 days	1	
14 days	1	1
18 days	2	1
20 days	1	
22 days	1	
Of indefinite duration	13 (Cases 1, 2, 4, 5, 6, 7, 9, 17, 29, 31, 38, 40, and the last of the silicin series)	3
Affording no evidence as to value of salicylic treatment	2 (Cases 26 and 32)	
Total . .	60	21

These figures do not seem to me at all unsatisfactory. In most of the cases which are set down as of indefinite duration it may be considered doubtful whether the value of the medicine was fully tested, on the ground that either too small a dose was given (Cases 2, 4, 6, 9, 38, 40) or that the persistence of pyrexia may be fairly attributed to the presence of pericarditis (Cases 1, 5, 7, and the last of the salicin series). And of the remaining two cases (29, 31) it is to be noted that in one the joint pains, and in the other the pyrexia rapidly fell.

Dr Greenhow, however, deals with his cases in a very different manner. He had, before beginning his observations, laid down the rule that "no patient should be put on the treatment with salicin or with salicylic acid, until he had been from twenty-four to thirty-six hours in the wards, and then only if it seemed clear that the illness was running an acute course." His object in giving these instructions was to exclude such cases as would, independently of any medicines, "improve rapidly after admission into the hospital, and become convalescent in three or four days." His plan, however, was not always quite strictly carried out, and the consequence is that he dismisses as valueless no fewer than twelve of those cases in which the subsidence of symptoms under treatment was most rapid. But surely the very question at issue is whether the administration of salicylic acid is, or is not, capable of shortening the duration of the attack. I certainly know of no evidence that so large a proportion of cases as this (in addition to an indefinite number of other cases withdrawn from treatment on the ground of their mildness) would ever get well in from one to four days without medicine. My own opinion, therefore, is that Dr Greenhow did in reality obtain good results from salicylic acid and salicin, but that in his anxiety to weigh strictly the therapeutical claims of these drugs he has dealt them scant justice. That the sixty cases which he submitted to treatment were of more than average severity is, I think, supported by the further fact that in about twenty-five of them signs of pericarditis were discovered either on admission, or within a day or two afterwards.

Prognosis and events.—It is only in exceptional cases that acute rheumatism proves fatal. Senator puts the average mortality at from 3 to 3·7 per cent. of those who are attacked. But at Guy's Hospital I think that it must have been much less than this. The number of deaths has varied very widely in different years. From 1855 to 1867 only *ten* cases altogether appear in our records of post-mortem examinations. Between 1868 and 1880 there were *forty-seven* fatal cases. The numbers in the several years were as follows: two in 1868, three in 1869, five in 1870, two in 1871, seven in 1872, two in 1873, seven in 1874, six in 1875, three in 1876, six in 1877, one in 1878, two in 1879, one in 1880.

In three of these fifty-seven cases, however, the fatal termination was really accidental, being due to stenosis of the mitral valve, which had itself resulted from previous attacks of acute rheumatism. In eighteen instances it was attributed to pericarditis, which had sometimes reached a very severe degree, the heart being covered with lymph from a quarter of an inch to one inch thick, and there being from ten to sixteen ounces of fluid, generally deeply stained with blood. In several of these cases there was also effusion into one or both of the pleural cavities; twice there was mediastinitis; in seven cases the muscular substance of the heart was obviously involved in the inflammatory process. Each of the complications in question may be supposed to have played a part in bringing about the patient's death; and indeed it is not improbable that myocarditis was really sometimes present when it was not noticed. On the other hand, although endocarditis existed in eleven of the eighteen cases, it was so slight that it could not be considered to have affected the issue. I can only find four instances in which a severe acute lesion of the valves was found as the direct result of acute rheumatism. One of them occurred in 1856 in a potman, aged twenty-five, who was in the hospital seven weeks, who had from the first a loud systolic murmur, and who did not convalesce when all his rheumatic pains had subsided; the mitral valve had upon it large vegetations of the size of filberts, and there were embolic plugs in the spleen and in the kidneys. Another case was that of a man, aged twenty-eight, in whom the cause of death, at the end of seven weeks, seems to have been empyema, accompanied by œdema of the lungs; upon the valve were two fungating masses, a quarter to a third of an inch in diameter. In a third instance the valve had hanging from it long vegetations like loose chordæ, and in a fourth the chordæ were themselves broken through; but each patient had had one or more rheumatic attacks before that which led to his death. There were three cases in which the fatal termination appeared to be due to pleurisy, bronchitis, or pneumonia; once it was traced to pulmonary embolism, and once to œdema of the larynx.

I have already alluded to a fatal case which occurred in 1880, and in which the autopsy afforded no very satisfactory explanation of the patient's death, so that Dr Goodhart was inclined to ascribe it to the administration of salicylic acid. When the question was discussed by the Clinical Society, Dr Bristowe threw out the suggestion that the "rheumatic poison" itself may sometimes act with sufficient intensity to destroy life. In our records I find several other cases, besides Dr Goodhart's, in which, although the death was set down to pericarditis, one may fairly doubt whether the inflammation was sufficiently severe to have produced the fatal result.

Hyperpyrexia.—I have still to mention the most important of all the causes of death in acute rheumatism, namely, high temperature attended with severe brain symptoms. That such symptoms sometimes develope themselves, and rapidly prove fatal, has long been known. By Sir Thomas Watson and Dr Latham it was thought that they were in some way dependent upon the cardiac complications of rheumatism; and cases were recorded which seemed to show that endocarditis, even without pericarditis, was capable of producing them. But in other instances the heart has been found free from all signs of inflammation. The existence of meningitis has also been disproved.

In 1867 Dr Ringer related three cases of rheumatic fever in which extremely high temperatures were observed, the thermometer having risen from 104° or 105° to 109·2° or even 110·8° Fahr. A German physician, Dr Kreuser, seems to have noticed the same fact in Würtemberg, about a year previously. Since then many similar instances have been recorded, both in England and abroad; at Guy's Hospital at least fourteen patients died of hyperpyrexia in acute rheumatism between 1870 and 1877. The most reasonable supposition seems to be that while the development of the high temperature is itself consequent upon an exhaustion of the heat-regulating centre in the bulb, the cerebral symptoms in their turn result from the action of the heated blood upon the brain. There are, indeed, other diseases in which the thermometer occasionally rises to a similar height before death; but this occurs only in very severe cases, and when the other symptoms cause grave anxiety.

But in acute rheumatism hyperpyrexia may suddenly bring to a fatal issue an illness which had seemed to be attended with little or no risk. One of Dr Ringer's patients was supposed to have recovered, and was about to leave the hospital on the next day, when cerebral symptoms set in, of which he died within two hours, his temperature being 110°. The first indication of the onset of hyperpyrexia is indeed very often that the patient loses his pains and finds that he can move all his limbs without suffering. He and his friends are naturally rejoiced at what seems to be a great improvement in his condition; but such an occurrence, unless the temperature falls at the same time, should instantly suggest to the physician a warning of impending danger, and should lead him to employ the thermometer at regular intervals of ten, twenty, or thirty minutes. Another point is that, as a rule, the skin ceases to perspire, and becomes dry to the touch; but in one case, observed at Guy's Hospital in 1874, there was still profuse sweating when the temperature had risen to 107·2°. Dr Wilson Fox says that the urine has sometimes been passed in excessive quantity and has been pale and of low specific gravity.

[Vide a paper by Dr T. D. Acland on "Salicylic Acid and Urea" in vol. viii of 'St Thomas's Hospital Reports.'—Ed.]

The cerebral symptoms, which rapidly supervene, vary in character in different cases. Sometimes the patient becomes drowsy, appears to fall asleep, and so passes into a state of unconsciousness, with contraction of the pupils, which ends in death. Sometimes he grows violently maniacal, jumping out of bed, and fighting with nurses and attendants. Sometimes he is seized with convulsions, or with tonic spasms and opisthotonos. His pulse becomes very rapid, from 140 to 186; towards the last it may cease to be perceptible. His breathing is much accelerated, and may have a sighing character. The face may exhibit a dusky purple flush, and the eyes may be deeply suffused; there may even be complete cyanosis.

The interval which elapses from the commencement of the hyperpyrexia to the fatal termination is very variable, as is well shown by an analysis of twenty-two cases made by Dr Wilson Fox. In one instance the thermometer rose from 103·5° to 109° in two hours; in two other instances a period of twenty-four hours passed before a similar point was reached. Even when the temperature is 107° or 108° the patient may now and then continue to live for some hours; and at 110° there may still be an interval of from one to two hours before the fatal termination. On the other hand, a woman died in Guy's Hospital with opisthotonos, after two hours' delirium, when the thermometer indicated only 103·9°. Another patient succumbed to delirium at a temperature of only 105·6°; he had been intemperate in his habits, and had had delirium tremens several times. Of course such cases cannot be spoken of as examples of hyperpyrexia; but I think that in the woman, at any rate, that condition may fairly be supposed to have been present in an early stage, and to have been cut short by fatal spasm before it had time to develop itself. She was taking salicylic acid, but I do not know in what doses.

It is perhaps worthy of notice that all but two of the patients who within the last few years have died of hyperpyrexia at Guy's Hospital, have been over twenty years of age; one was a youth of eighteen, another a girl of nineteen. I have already remarked that this complication often arises in cases which had appeared to be mild, and to be running a favourable course. Dr Fox seems to think it is more frequent in first attacks of rheumatic fever than in subsequent ones; but among the twenty-two instances collected by him there were five in which the patient had had the disease before and five in which the point was doubtful.

The treatment adopted before the setting in of serious symptoms, has varied widely; sometimes alkalies had been given, sometimes colchicum, sometimes iodide of potassium. Two of Dr Fox's patients were taking the tincture of iron in large doses. I must confess that when so many cases of hyperpyrexia occurred in Guy's Hospital from 1871 to 1877, I acquired a notion that their occurrence was not altogether unconnected with the fact that the tincture of iron, quinine, bark, and ammonia were at that time so commonly administered to patients suffering from acute rheumatism. It was also usual to envelop all the affected joints in thick masses of cotton wool. There can of course be no question that the profuse sweating which accompanies the disease, whether advantageous or harmful to the patients in other respects, must tend greatly to keep down the pyrexia. And I think that for the future one should be careful not to give medicines which may check perspiration without lowering temperature, nor to use local applications which may prevent evaporation.

How far the administration of salicylic acid or salicin affects the liability of the patient to hyperpyrexia is a question of extreme importance. This complication arose in two of the 355 cases which I collected from the records of Guy's Hospital as having been treated by these remedies between 1876 and 1880. But in one instance it was only twenty-four hours after the first dose of twenty grains of salicylic acid that the temperature reached 106·4°; and in the other it is not stated that the patient was taking the medicine when the hyperpyrexia developed itself, five days after admission. As yet I know of no instance in which the temperature had in the usual manner been falling under salicylic acid, and has then suddenly swept upwards, beyond all ordinary limits. But if the remedy in any case fails to bring down the pyrexia, the proper inference is, I think, that it is being administered in an insufficient quantity; at any rate large doses sometimes succeed perfectly when smaller ones have failed. Dr Greenhow speaks of hyperpyrexia as having developed itself in two of his cases. But in one of them the patient had been taking but fifteen grains of the salicylate of soda at intervals of six hours; and even this seems to have been discontinued the night before the temperature began to rise to an alarming height. In the other case the thermometer indicated 105·8° at a time when twenty grains of salicylate were being taken; but the progress upwards was by no means rapid, and no cerebral symptoms were manifested; it is therefore quite possible that a fall might have afterwards taken place if the treatment had not been interrupted.

The discovery of the real cause of the supervention of dangerous cerebral symptoms in rheumatism was necessarily soon followed by the employment of active antipyretic treatment, in the hope of averting the fatal issue. Hitherto the administration of salicylic acid, or of quinine, has resulted only in failure, at least in cases in which the temperature has been rising rapidly and has reached a great height. But wonderful success has sometimes been attained by the application of cold.

The first instance in which such a procedure brought about the recovery of a patient affected with hyperpyrexia in acute rheumatism seems to have been recorded by Dr Meding in the 'Arch. f. Heilkunde' for 1870; the temperature was 108·6°; he employed cold affusion and enemata of iced water. In the following year Dr Wilson Fox had two cases, the publication of which drew the attention of the whole profession in this country to the subject. The first occurred in a woman, aged forty-nine, who was in the fourteenth day of her illness, and who had been in University College Hospital five days, when her temperature began to move rather quickly upwards. At 3 p.m. it was 105°; at 6, 106·4°; at 8·5, 107·1°; at 9·15, 108·4°; at 9·50, 109·1°. She had then been completely unconscious for a considerable time, her pulse was imperceptible, her face was cyanotic, and she appeared to be drawing the few last irregular gasping respirations which commonly precede death. There had been delay in preparing a bath, into which, at a temperature of 96°, Dr Fox had intended to put her when her temperature should have been 107°. However, she was lifted into it at 9·50, and five minutes later the temperature in the rectum was found to be 110°. With admirable courage, Dr Fox sent for some ice; two large lumps were placed, one on her chest, and another on her abdomen; a bag filled with it was tied down the length of her spine; two assistants baled the warmer water out of the bath; and two others poured iced water over her as fast as the pails could be filled. The temperature in the rectum gradually fell until at 10·25 it was 106·2°. The pulse now became perceptible, and some slight signs of consciousness were manifested. At 10.35, the temperature in the rectum was 103·6°, and she was removed from the bath. At 10·55 the temperature in the rectum was 100·6°; she could speak, but her consciousness was still very imperfect. The bath had to be repeated on the following morning, but she finally recovered. Dr Fox's second case was that of a man, aged thirty-six, in whom on the sixth day after his admission (the seventeenth of his disease) the temperature rose to 107°, having before been always below 104·5°. He showed signs of pericardial effusion, and also of inflammation at the bases of both lungs; he had cough and expectorated a thin mucoid fluid stained with blood. This did not prevent Dr Fox from having him placed in a bath at 89° for twenty-five minutes, during which time it was cooled down to 86°. The temperature in his rectum fell from 107·3° to 103·1°; and after removal from the bath it became normal. He ultimately got well, after having had eight baths in all.

During the time which has passed since Dr Fox recorded his cases, the treatment of hyperpyrexia by cold has been repeatedly adopted, and with very satisfactory results. One of the most striking instances that I know of occurred in the person of one of my pupils and friends, who in 1875 had a very severe attack of acute rheumatism, during the course of which his temperature on twenty-six occasions, from the ninth to the twenty-fifth day of March, rose to a point between 105° and 107·2°, being each time brought down by immersion in an iced bath. He recovered, and is now engaged in medical practice. Full particulars of this case may be found in a paper in the 'Liverpool Medical Reports' for 1876 by Mr F. T. Paul, who was house physician at Guy's Hospital at the time, and who carried out the treatment for me with unwearied patience and determination.

But, unfortunately, even when the bath is perfectly successful in lowering the patient's temperature, it does not always restore his consciousness, still less save his life. Thus Mr Paul records the case of a man, aged thirty-two, one of the porters in Guy's hospital, who, after a week's illness with rheumatic fever, became extremely delirious and then comatose, and was found at 9 p.m. with a temperature of 108·8° in the axilla. As he lived out of the hospital there was a delay of at least an hour before a bath could be procured. When he was put into it his temperature was 110·9°, and he was violently purged. The bath was at 90°, and he was kept in it for thirty-five minutes, during which time it was reduced to 66° by cold water. His temperature on removal was 106·6°; he was still perfectly insensible, with contracted pupils, and with noisy and rapid breathing. Half an hour later the temperature in the rectum was 101·3°, and an hour after the bath it was 99·3°. Subsequently it rose slightly, but it never reached 103°. He died in the afternoon of the following day, the only change in his condition being that the contracted state of the pupils gave way to wide dilatation.

Another instance, which may also be found in Mr. Paul's paper, is that of a woman who died after having had twelve baths, during a period of nine days; for the last two or three days mucous râles were audible widely over the chest; but at the autopsy nothing was found, except a little bronchopneumonia at the bases of the lungs, and some mucus in the tubes. We have had five other cases at Guy's Hospital which ended fatally, notwithstanding that the hyperpyrexia had been overcome by baths; in only one of them did the post-mortem examination reveal an adequate cause of death in a severe pleurisy with pericarditis. Such patients seem generally to sink by failure of the circulation. Indeed, in both of Dr Wilson Fox's successful cases it was deemed necessary to give large quantities of brandy after the baths, and also to apply hot bottles to the feet and warmth to the back; his first patient took six ounces of brandy within an hour, at the time of the first bath.

In all probability the best method of averting collapse after hyperpyrexia is to have recourse to a bath at the sufficiently early period, before the heart and the tissues generally have been too much damaged by the heat. Moreover, when the bath is too long delayed there is always some risk of death during immersion, which has happened to two patients at Guy's Hospital, once in 1874, and again in 1877. The rule I think, should be that when the temperature is rising for the first time to a dangerous height, it should be allowed to reach 107°, but not to go beyond it before the bath is used. All the necessary preparations, however, should be made as soon as the thermometer indicates 105°, and the supervention of convulsions may make it necessary to immerse the patient at that temperature, or at 106°. Subsequently, the bath should be had recourse to as often as the temperature rises to 106°, but not below 105·5°. It is best to let the water have a temperature of 90° at the time when the patient is immersed; if it be much colder than this he is likely to shiver and complain; whereas when it is from 90° to 110° he finds it exceedingly pleasant, so that he will often afterwards beg to have the bath repeated, before it seems to be absolutely required. He should be lowered into the water upon a sheet. As soon as this has been done the temperature of the bath should be reduced to 75° or even to a still lower point, by the addition of ice, which is more convenient than cold water, because it occupies less space, so that no baling out is required. The patient should not be left in the water after the temperature in his rectum has fallen to 102°, as it will continue to go down subsequent to his removal. When he has been lifted back upon the bed he should be lightly covered with a blanket.

Diagnosis.—Acute rheumatism is, as a rule, very easy to recognise. But there are not a few diseases which may be mistaken for it by a careless observer, and in some exceptional instances even one who takes pains may be unable to decide. It should, I think, be a cardinal rule to ascertain that at least one of the joints is really swollen, and, if possible, that it contains fluid exudation in its interior, before one commits oneself to an opinion.

Not long ago, I was called into the hospital one Sunday by the house physician to see a girl who had been just admitted for rheumatic fever, and whose temperature was very high. The peculiarity of her case was that she was covered all over with a bright scarlet rash. She had had acute rheumatism on a former occasion, and perhaps that circumstance threw me off my guard; but it never occurred to me to suspect the real nature of her illness, which became apparent on the following day, when the papular eruption of *smallpox* was found upon her. I have seen several instances in which the pains in the limbs produced by the growth of *multiple sarcomata* in the body, especially about the vertebræ, so as to affect the spinal nerves near their origins, have been supposed to be due to acute or to subacute rheumatism; whether joint affections are ever really present in such cases as those referred to at p. 94 of vol. i, I do not yet know.* So, again, with *ulcerative endocarditis.* This disease has been described as sometimes complicating acute rheumatism. Cases which were thus interpreted are recorded by Sir Thomas Watson. But, as he does not positively state that the joints were swollen, and as it is now known that—in primary ulcerative endocarditis—severe pains in the limbs and in other parts are often complained of, I think it is permissible to feel some doubt as to whether the rheumatism was really present. Again, it formerly often happened that affections of the spinal cord were in their early stages regarded as rheumatic on account of the pains to which they gave rise, and that which first showed a mistake to have been committed was the supervention of paralysis. But it must not be forgotten that in some spinal cases, and notably in those of locomotor ataxy, the joints do actually suffer. The same remark applies also to *syphilis*, the effects of which are not infrequently set down to rheumatism, as in a case that was lately brought to me. Lastly, *scorbutus* and *hæmophilia* must be mentioned as diseases with regard to which it is by no means difficult to make a similar blunder.

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* [In a case of multiple sarcomata of the skin, secondary to a deeply-seated growth in the pelvis, under my care, there were pains and swelling about the joints which I mistook for rheumatism, and when the sarcomata appeared I supposed them to be purpuric erythema —peliosis rheumatica.—Ed.]

In most of these cases the joint affections, if present at all, are such as would correspond with a mild or subacute attack of rheumatism, rather than with the more typical forms of it. But there are other instances in which with high fever and with obvious intense inflammation of several articulations, a diagnosis is very far from being easy. I have several times been for some days unable to say whether a patient had *gout* or acute rheumatism, so ambiguous were the symptoms; and in other cases I have felt confident that the disease was gout, when it has turned out acute rheumatism—or *vice versâ*.

But perhaps a more common mistake is that of setting down for acute rheumatism one of those forms of *pyæmia* which come under the observation of physicians, because they are not secondary to any wound or any obvious injury. In most of the cases in question there is, as a primary lesion, *osteomyelitis*, of one or more of the long bones. This is often easily recognised by anyone who will carefully examine the patient. The corresponding part of the limb may be greatly swollen, and very hot and painful; and there may be a distinct history of its having been injured in a fall or by a blow.

But sometimes matters are very different. In 1879 Dr Goodhart made an autopsy in the case of a boy, aged sixteen, who had been lying for five days in a medical ward in a typhoid condition. He had a systolic murmur; his temperature had been 103·8° on admission, his pulse 132, his respirations 48. He had been attacked with pains and with chilliness, five days before his admission. Numerous abscesses were found in the lungs and in the kidneys. Evidently, therefore, the case was one of pyæmia; but it was not until after nearly every other bone had been examined that Dr Goodhart discovered suppuration beneath the periosteum of the lower end of the right fibula. On the tricuspid valve there was a vegetation of the size of a pea, with a little ulcer beneath, which had torn through some of the chordæ. Acute osteomyelitis cannot, indeed, be set down without further question as the cause of pyæmia in every instance in which it is found. Dr Moxon says that he once saw such an affection in a case of pyæmia which arose from a carbuncle in the back, so that the bone lesion was there an effect, and not the cause, of the blood-poisoning. But I nevertheless think that, as a rule, it is the cause, and that if one could examine the cancellous tissue of every bone in the body, very few cases would have to be set down as instances of so-called "idiopathic" pyæmia. How it happens that an inflammatory process developed in the interior of solid structures should acquire the property of infecting distant parts I cannot say. In vol. xxiii of the 'Guy's Hospital Reports' Mr Howse has thrown out the suggestion that the poison of one of the exanthemata circulating in the body may perhaps serve to convert a simple into an infective osteo-myelitis; but this hypothesis seems to me to have but little probability in its favour.

How closely pyæmia may resemble acute rheumatism is well shown by a case which occurred to Dr Moxon in Guy's Hospital in 1877. A patient was admitted who had nine days before been attacked with headache, sickness, and rigors, and in whom these symptoms were followed by profuse sweating and by pains in the joints. Salicylic acid was prescribed, but the temperature rose and delirium set in, so that cold baths were employed on several occasions. The question whether the disease could be pyæmia was formally discussed, and negatived in favour of the diagnosis of acute rheumatism. No complaint of the thigh, as being more painful than other parts, was made when he was being moved into or out of the bath. Yet at the autopsy I found not only that there was inflammation of the cancellous tissue of the lower part of the shaft of the right femur and of the adjacent epiphysis, but also that the bone was denuded of its periosteum, that there was a large collection of dirty pus beneath the muscles. There was besides suppuration about the shoulder, and there were pyæmic abscesses in the lungs and in the heart. In that instance the right knee-joint was not suppurating, but contained much serum and many loose masses of lymph: and we have had three or four other cases of this form of pyæmia, in which at least some of the joints that had been noticed to be swollen during life have been in a similar state. The fluid in them was described as "an excess of synovia," or as "curdy synovia," or as "an excess of serum with lymph." It therefore seems doubtful how far reliance can be placed on one point which I had supposed to afford an important distinction between acute rheumatism and pyæmia, namely, that in the former disease the pain and the swelling are so apt to fly about, leaving one joint, and after a few hours, attacking another.

In the case of a boy who died of ordinary pyæmia in a surgical ward in 1870 I find it noted that there was at first fugacious pains in the joints. It is worthy of notice that almost all the instances that have occurred at Guy's Hospital within the last few years of pyæmia from osteomyelitis have been in boys or young men between ten and twenty years of age; but I have seen such an affection in an infant. As a rule, the diagnosis is rendered comparatively easy by the severe constitutional disturbance which is present, by the presence of rigors, by the skin being dry instead of sweating; the joints, too, show a deeper blush, and are more frequently hot. One very important question is as to the significance of certain eruptions which are sometimes associated with pyæmia. I am not referring merely to the scarlatiniform rash which is comparatively common in surgical wards, and which has now been proved to be really scarlet fever. There are cases in which the skin presents appearances of a still more remarkable kind. Thus in 1861 a boy of thirteen was admitted into Guy's Hospital who had for a week been treated for rheumatic fever, but whose disease was at once recognised to be pyæmia, there being a large abscess of the thigh. Towards the last, his body became covered with "a purplish rash, resembling the mottled rash of typhus, partly consisting of petechiæ (probably fleabites), partly of papules which became vesicular at the apices and slightly scabbed." In another patient, in 1874, pustules are said to have appeared on the back and on the abdomen two days before death. So, again, eruptions are mentioned in two instances of "spontaneous pyæmia" collected by the Pathological Society's Committee in 1879; in one case it was at first like impetigo, but afterwards consisted of "hard inflamed lumps, the size of peas, on each leg, and of suppurating, red, raised patches on one forearm;" in the second case there was "a vesicular eruption, in some places pustular, on the fingers and toes and forehead."

In regard to all such cases, however, it is necessary to be careful not to overlook the presence of *glanders*. That disease is itself sometimes mistaken for acute rheumatism at its commencement, and, as I have already pointed out (vol. i, p. 317), pyæmia is very commonly associated with it.

Another class of cases which are very likely to be set down to acute rheumatism are those in which *pyæmia* results *from gonorrhœa.* I shall have to refer to this question again; but I may here mention what was probably a case of this affection in a female. In 1872, a young woman was admitted into Guy's Hospital with what was supposed to be rheumatic fever; an hour later she was delivered of a child which survived for some days; she was now seen to be suffering from pyæmia, and four days afterwards she died. At the autopsy an abscess was found in the subserous tissue near the right ovary, and there were softening thrombi in the adjacent veins; but it was thought that these lesions were themselves secondary, and that the starting-point of the disease was a vaginal discharge from which she had been suffering, and which was probably of a gonorrhœal character.

But the most remarkable case of pyæmia that I ever saw was one which occurred in 1874 in a boy, aged four and a half, who was admitted for a well-defined tumour of cartilaginous hardness, under the skin at the upper margin of the left orbit. The surgeon talked of excising it, but it was discovered that the child was very feverish, and death quickly followed. The supposed growth was then found by Dr Goodhart to consist of orbital periosteum infiltrated with pus; and the inflammatory process was traced by continuity to the pterygoid region, and so along the inferior dental canal to the left first molar, which was in a state of caries. In that instance there was no affection of the joints, but the lungs contained pyæmic abscesses.

In ordinary surgical practice it of course seldom happens that pyæmia is mistaken for acute rheumatism. But sometimes the converse blunder is committed, and when a rheumatic affection of the joints arises in a person who has been recently operated on or who is suffering from an accident, pyæmia may at first be feared.

[*Rheumatic nodules.*—An additional concomitant of rheumatism, of both pathological and diagnostic interest, has been lately discovered—small, subcutaneous, fibrous nodules, usually but not always in the neighbourhood of joints, often upon prominent points of bone, like the olecranon, the tibia, and the acromion. They were described by Meynet, of Lyons, in 1875, by Rehu in Germany (1878), by Hirschsprung in Denmark (1881); and a full account of them by Dr Barlow and Dr Warner will be found in the 'Transactions of the International Congress of 1881,' vol. iii, p. 116, with twenty-seven well-observed cases.

Numerous cases have since been recorded by Dr Cavafy ('Path. Trans.,' xxxiv, p. 41), Drs Duckworth, Money, Drewitt, Stephen Mackenzie and Fowler, in the 'Transactions' of the Pathological Society for 1883 and of the Clinical Society for 1883-4.

In microscopical structure and in relation to acute rheumatism the numerous cases hitherto reported agree.—Ed.]

ARTHRITIS DEFORMANS

Nomenclature—Relation to gout and to rheumatism—Ætiology—Anatomy of the joints—Symptoms and course—Acute cases—Resulting deformity—Involution and recovery—Treatment by drugs and external applications, by diet and climate.

From the beginning of this century it has been known that beside Gout and acute Rheumatism, and apart from the so-called subacute and chronic forms of the latter disease, there is a third affection to which the joints are liable, and by which a large number of them may be crippled in the same patient, either at the same time, or in succession. This affection had been noticed as a modification of rheumatism by Sydenham; but the first description of it as an independent malady was that which Dr Haygarth gave in 1805, under the name of "Nodosity of the Joints." It has since received various appellations, among which may be mentioned "rheumatoid arthritis" (Garrod), "rheumatism noueux" (Trousseau), "arthritis pauperum," "chronic rheumatic arthritis" (Adams), "osteo-arthritis," "malum articulorum senile," "arthrite sèche" (Cruveilhier), "arthritis deformans." The last of these is the name by which it is most commonly known on the Continent, and is perhaps the most satisfactory. In popular language the term "rheumatic gout" is generally applied to it. This, however, is much to be deprecated, since an inevitable consequence is that the distinction between it and chronic gout becomes lost or ignored.

There are, indeed, still some observers who maintain that all these joint diseases, including even acute rheumatism, are closely related to one another, and in common depend upon what is termed an *arthritic diathesis.* This view is held by Mr Hutchinson, who thinks that he can identify an "arthritic" iritis, and even an "arthritic" pneumonia, by their recurring again and again in the same individual, and being each time recovered from. But it seems to me that a large part of the evidence which he adduces in support of his doctrine is rendered valueless by the uncertainty which clings to the use of this very term "rheumatic gout." He brings forward instances in which different members of the same family, belonging perhaps to successive generations, are said to have suffered from different "arthritic" affections; but I doubt whether one can attach any significance to a statement that a mother, or an uncle, or a grandfather had "rheumatic gout," or "chronic rheumatism," or even "gout," unless we have a more detailed account of the case than can generally be obtained. It is, indeed, certain that the lesions characteristic of gout and those which belong to arthritis deformans are sometimes found in different joints of the same person;* but I am not sure whether this occurs more frequently than might be expected as a mere coincidence. Between 1874 and 1879 four examples of such an association were met with by Dr Goodhart or by myself in the post-mortem room of Guy's Hospital. Three of the patients were men, aged fifty-two, fifty-four, and sixty-two respectively. The fourth was a woman, aged thirty-six; and it is an exceedingly interesting fact that she was said to have had rheumatic fever at the age of twelve, which in all probability was really the case, for she died of mitral stenosis. In each instance the great toe-joints contained urate of soda, showing that gout had been present, while the lesions indicative of arthritis deformans were found in the knees or in the hip-joints; once the left knee showed both kinds of change, the opposite knee being affected with arthritis deformans alone, and the two great toes with gout alone. I therefore feel rather disposed to leave it for the present an open question whether there may not really be some relationship between these diseases. That acute rheumatism sometimes passes on into arthritis deformans seems to be certain. But then one must bear in mind that the latter affection is probably identical, from a merely anatomical point of view, with joint affections that may arise from injury, or in various other ways. Thus Mr Hutchinson in the 'Med. Times and Gaz.' for 1881, mentions that in a young woman whose thigh was amputated by Mr McCarthy for a myeloid growth in the tibia, he observed small lips on the edges of the condyles of the femur. Now, this appearance, as I shall presently point out, constitutes one of the most marked indications of arthritis deformans; but in that case it was doubtless the result of irritation caused by the proximity of the tumour.

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* [And still more rarely, even in the same joint.—Ed.]

Ætiology.—So far as the causes of arthritis deformans are known, they appear to differ altogether from those of gout, and to some extent also from those of acute rheumatism. Dr Garrod, in 'Reynolds' System,' says that after looking over a large number of cases he was unable to find much evidence of its being transmitted by inheritance; one member of a large family not infrequently suffers severely from it, while the rest remain free. Again, it does not seem to be produced by indulgence in rich food or in alcoholic stimulants. It is apt to occur rather in weakly, ill-fed persons, who are exhausted by rapid child-bearing, by prolonged lactation, by grief, or mental anxiety. Some pathologists have surmised that it depends upon a morbid condition of the nervous centres, basing their opinion mainly upon the fact that a similar joint-disease arises in locomotor ataxy.

On the other hand, Dr Garrod believes that the subjects of the tubercular diathesis are especially liable to be affected by it; as also are "individuals of weak frame, whose circulation is languid, and whose extremities are habitually cold." Among twenty-nine cases collected by Dr Pye-Smith for a paper in vol. xix of the 'Guy's Hospital Reports' (1874), there were three in which phthisis was also present. Dr Ord, in the 'Transactions of the Clinical Society' for 1877, has brought out, more prominently than other observers, its relation to dysmenorrhœa and to "ovario-uterine provocation." In one of his patients it was regularly developed paroxysmally just before, throughout, and for a short time after each menstrual period, and underwent no less regular remissions in the intervals. He even noticed that in three cases in which the joint affection was limited to, or began and remained excessive in, one side of the body, the ovary on the same side was painful and tender.

In some cases, as Haygarth remarked, arthritis deformans develops itself at the climacteric age. It is, however, by no means confined to the female sex, or to any one period of life. Dr Garrod says that he has seen it in its worst form in children of ten or twelve years, and he has also met with instances in which it began in very old people above seventy years of age. I remember what appeared to be a well-marked instance in a lad of eighteen; but I ought perhaps to add that I do not know of any case, in a young subject, in which the identity of the affection with that which occurs at later ages had been verified by an autopsy. According to Senator, 'Ziemssen's Cyclopædia,' vol. xiii (p. 141), the characters of arthritis deformans differ somewhat according to the sex and age of the patient; the cases in which it begins in the hands and feet, subsequently spreads to the larger joints, almost always occur in women; men are more liable to have it in the hip or in the shoulder, before any other parts are affected. He says, too, that the latter variety of the disease is especially frequent at an advanced period of life, that it is seen indifferently in the rich and in the poor, that it bears little or no relation to depression of the general health or to exposure to cold and wet; whereas the former variety is particularly common in those who live in damp houses, and where the air is moist and chilly. A similar distinction has been drawn by Dr Pye-Smith. Indeed, as Mr Hutchinson points out, the change in the affected joints is not quite the same at different ages; in persons under middle age outgrowths of bone, which we shall see to constitute a striking feature of the disease in old people, are rare; and if present they are usually small.

A point on which Senator insists is that the parts most apt to be attacked are such as have been most used; as, for instance, the fingers and the wrists of watchmakers, and of women who have worked hard with the needle, or at knitting. In connexion with this it may be worth while to mention that, as Dr Pye-Smith remarks, a similar affection occurs in the feet of horses, perhaps as the result of over-work; but it has also been noticed in the vertebræ of whales by Prof. Struthers. With regard to the geographical distribution of arthritis deformans in man I believe that no accurate observations have yet been made. But Trousseau, after stating that it is "a disease of rare occurrence" (in Paris?), remarks that in "certain damp countries it is so common as to be almost endemic." A circumstance of some interest is that signs of it have been detected by Della Chiaje in bones taken from Pompeii, and by Lebert in bones from the catacombs of Paris, so that the disease is clearly not of modern origin, although it has only been distinguished of late years.*

Anatomy.—At the commencement of arthritis deformans the morbid process is often limited to a very small area of one or both of the articular cartilages of a joint. Sometimes it begins at the centre of the cartilage, sometimes (as in several specimens exhibited to the Pathological Society by Mr Hutchinson in 1872) it begins round the margin, and spreads irregularly inwards. The cartilage first becomes soft and velvetlike in appearance; afterwards it ulcerates, so that there is formed in it a cavity, with a more or less sharp edge, in the floor of which the bone may be exposed. Histologically the change consists in a proliferation of the cartilage-capsules, so that each becomes replaced by from eight to twenty large elements; the matrix at the same time splits into fibres in a direction perpendicularly to the articular surface. Presently the enlarged capsules rupture into the joint cavity: the fibres then remain for a time as shaggy projections, until ultimately they too disappear. The denuded bone sometimes exhibits an open cancellous tissue, but it is more often converted into a very hard compact substance, or (to employ the usual term) undergoes *eburnation*. When, after the removal of the whole of the articular cartilages, the osseous surfaces everywhere come into apposition, with no soft material between them, they become scored and fluted with parallel grooves and ridges, corresponding in direction with some particular line of movement, to which they are henceforth restricted. On the other hand, the texture of the subjacent part of the bone becomes wasted, loaded with fat, and undergoes gradual absorption. Thus the neck of a femur, for example, may be gradually shortened, until what represents the head lies deeply in a hollow between the two trochanters. Intra-articular fibro-cartilages, as in the temporo-maxillary joints, resist the disease no better than the cartilages which cover bones. So also the ligamentum teres becomes lost when the hip is affected. And in the shoulder the long head of the biceps very constantly disappears; it seems to undergo fusion with the capsular ligament of the articulation, so that it cannot be traced upwards beyond a certain point, and yet no definite free end can be found. These various changes are attended with a gradual loss of mobility, and at length the spaces between the bones may become filled up with fibrous tissue, so that a condition of ancylosis is established.

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* [There is no question that Arthritis deformans is more common in Ireland than in England or Scotland, and it is more frequent among agricultural labourers than in towns. For an account of a specimen from a Roman tomb by Dr Norman Moore, see 'Path. Trans.,' vol. xxxiv.—Ed.]

Formative changes indeed are generally, though not always, associated with the destructive process from a very early period. From the edges of the articular cartilages there arise a series of nodulated outgrowths—or "ecchondroses," as they are termed—which form a kind of raised lip or border. These are at first small, but they afterwards increase in size; and soon lime-salts are deposited in them, so that they become converted into so many bony plates or masses, which may extend far into the capsular ligament, or into the tendons round the joint, and may even unite to form a complete osseous shell. In like manner, outgrowths from the edges of the bodies of the vertebræ often cohere together across the intervertebral discs, so as to constitute a number of bony splints, which may immoveably fix a large part of the spinal column.

Probably one should regard as a modification of the bony outgrowths of arthritis deformans, an affection which was long ago described by Heberden under the name of "Digitorum Nodi," and which, as he said, consists of "little hard knobs, about the size of a small pea, situated upon the fingers, particularly a little below the top, near the joint." They continue for life, and although they give rise to no pain, they are often brought under one's notice by ladies, because they are unsightly, and because of a fear that they may indicate the approach of gout. It is remarkable that a solitary excrescence should thus arise, and remain for years single, if it is really related to arthritis deformans; but, in one patient, I saw another finger-joint after a long time affected with chronic hydrarthrosis, so that in that instance there seemed to be no doubt about the matter.

In many cases the synovial membrane of the affected joints shows obvious changes. It is more or less thickened, especially near its lines of attachment to the bones. The folds, which project from it into the joint-cavity, are greatly enlarged and very vascular; they often form long villous processes, with numerous bodies like melon-seeds hanging from them.

Symptoms.—The early stages of arthritis deformans may be attended with no subjective symptoms whatever. This fact is well illustrated by one of Mr Hutchinson's cases in the 'Pathological Transactions;' a man, aged forty-one, had his thigh amputated for destructive inflammation of the knee, it not being known that any other joints were affected; when the limb was dissected, ulceration of cartilage was found in every articulation of the foot, except the distal joints of three toes. So, again, the same writer speaks of having frequently been able to detect the presence of a projecting lip round the articular cartilage of the lower end of the femur, in persons who are not aware that the knee had ever suffered. He places himself in front of the patient, puts the finger-ends of one hand flat upon one condyle, and those of the other hand upon the other condyle, and then directs him to bend and extend the joint slowly several times in succession. In this way, he says, the edges can be easily found, and the degree of their elevation estimated. He admits, however, that practice is required to prevent one's mistaking for a morbid condition a ridge which normally exists at the same spot in many healthy persons.

Thus it is probable that when a patient begins to complain of pain and stiffness in one or more of his joints, the disease has often been already present for a considerable time. The pain is not generally constant; it "comes and goes," sometimes without obvious cause, sometimes in apparent relation to changes of weather. In some patients it is worse when the limbs are warm, in others when they are cold; in some by night, in others by day. It is very apt to be brought on by the use of the part; in the hip, for example, by walking even a short distance; in the shoulder, by carrying anything, however small, in the hand. The stiffness, however, is apt to be more noticeable after rest, as when the patient first attempts to get out of bed in the morning. A sense of weakness and of distressing fatigue may be as marked a symptom as pain; and it must be noticed that there is often far more wasting of the muscles than seems to be accounted for by mere disuse of the limbs; the thenar and the hypothenar eminences, for example, may be so hollowed that one almost hesitates whether the case may not be one of progressive muscular atrophy. In some cases the pain has a shooting character, so as to resemble neuralgia.

Another very characteristic symptom, which, however, is not present during the early stage of the disease, is creaking or grating, which accompanies the movements of the affected joints; not only can it be plainly felt or even heard by the patient, but one can easily recognise it for oneself by grasping the part with one's hand.

But in some cases—probably at the very commencement of the morbid process—arthritis deformans sets in from the first with well-marked symptoms: it produces swelling, heat, and even perhaps redness of the affected joints, and is attended with more or less pyrexia. Indeed, Dr Garrod describes an *acute variety* of the disease, which, he says, very closely resembles ordinary acute rheumatism, differing only in the greater length of the paroxysm, in the absence of profuse sweating, and in its having no tendency to attack the heart. Trousseau, however, states that in four out of nine autopsies of "nodular rheumatism" made at the Salpêtrière by Cornil, pericarditis was present;* and Mr Hutchinson declares that it sometimes gives rise to paroxysms as short, and as definite, as those of gout itself. Moreover, in one of Dr Ord's cases I find a note that the temperature one evening rose to 102·8°. But, as Dr Garrod remarks, the difficulty in most instances of supposed acute arthritis deformans is to determine whether the disease may not really in the first instance have been acute rheumatism, and subsequently have passed into the other affection, as time went on. There is no doubt that osteo-arthritis is very often attended with effusion of fluid into the affected joints, and that the designation of *arthritis sicca*, which has sometimes been applied to it, is altogether inappropriate. Mr Hutchinson even maintains that almost all cases of so-called *hydrops articuli* really belong to this disease. Analyses of fluid from the hip-joint in arthritis deformans have been made by Hoppe-Seyler, and recorded in 'Virchow's Archiv' for 1872; he found in it a proportion of mucin which greatly exceeded that contained in normal synovia.

In other cases hæmorrhage takes place into some of the affected joints. Thus in 1875 Dr Goodhart, in examining the body of a man aged fifty, who had been admitted for arthritis deformans, but had died of hernia, found that each ankle contained several drachms of liquid blood, and that there was also blood in both knees, the synovial membrane being greatly swollen, with rounded vascular œdematous fingers; all these joints, as well as the hips which contained no blood, showed the characteristic changes of arthritis deformans. (See also 'Path Trans.,' vol. xxvi, p. 162.)

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* [It is incredible that this could be the result of a disease which rarely, if ever, produces cardiac lesions: and on looking into the cases, it is, I think, at least probable that chronic cirrhosis of the kidneys was the real cause of the pericarditis and death of some of these old cases of arthritis.—Ed.]

Among the less common symptoms of arthritis deformans must be mentioned the presence of fibrous nodules at a distance from joints, as, for instance, among the muscles of the arms or of the forearms. These are distinct from nodules of true rheumatism mentioned above (p. 552). I lately saw an old lady, aged seventy-one, who for about three months had complained of a curious affection of the tongue and cheeks, which perhaps belonged to this disease, inasmuch as she also had hydrarthrosis of each shoulder-joint, and a less marked affection of her knees. The tongue was uniformly enlarged, and had a peculiar firm fleshy consistency, without being at all indurated; its surface was rather smoother than natural. At each corner of the mouth there was a button-like mass which extended outwards for some distance into the substance of the cheek, and on which the mucous membrane adhered more closely than elsewhere to the subjacent tissues.

Dr Garrod mentions, as "irregular manifestations of the disease, sclerotitis, inflammation of the internal ear, and hoarseness, with a dry cough." But it seems to be not improbable that the aural and the laryngeal affections may have their seat in the small joints which are included within the organs of hearing and of speech.

Events.—At an advanced stage of arthritis deformans the deformity produced by it is generally very characteristic. The joints as a rule, are fixed in a flexed position. But the wrists are commonly extended. The fingers almost always lie at an angle with the rest of the hand, being deflected to the ulnar side, so that the knuckle of the forefinger projects strongly outwards towards the thumb. Some of the phalangeal joints are sometimes over-extended, so as to be concave on the dorsal aspect; this feature of the disease was noted by Sydenham. The degree of enlargement is very variable. Sometimes each articulation forms a bulbous swelling; sometimes the ends of the bones are almost of normal size. The patient is often completely crippled, unable to dress or to undress, to carry food to the mouth, or even to hold a paper in the hand. Yet the disease seems to have little or no tendency to shorten his life, its duration from first to last being perhaps ten, twenty, or even thirty years. Sometimes exhaustion by pain seems to be partly concerned in bringing his sufferings to a close; but more often death, when it arrives, is directly attributable to some intercurrent affection.

But it must not be supposed that the prognosis is always unfavourable, nor that all patients who become the subjects of arthritis deformans, at least in its slighter forms, necessarily continue to suffer from it for the rest of their lives. I remember among my friends an old gentleman who, some years ago, when he must have been nearly seventy, was unable during one winter to move the right shoulder, so that he had to be shaved by his servant, and required help in many other ways. During the following summer he became free from the complaint, and it has never since returned. Dr Ord speaks of more than one of his patients as having regained a fair state of health when menstruation became normal; but he does not say that joints which had been enlarged ever returned to their natural size.

Treatment.—The medicines most serviceable in arthritis deformans are iodides, arsenic, guaiacum, cod-liver oil, and actæa racemosa. Trousseau, following Lasègue, recommends the tincture of iodine in doses of ♏iij to xv. Iodide of potassium is more often prescribed in England. Dr Garrod says that it is especially likely to be useful when warmth augments the pain. He also speaks highly of the syrup. ferri iodidi, of which he orders twenty to thirty minims three times a day. Arsenic sometimes does great good, as I myself have once seen; it may be given in a small dose, but must be continued for a long time; it often fails entirely. Of the actæa racemosa, Dr Ringer says that it has yielded him very satisfactory results, and that it is most useful when the pain is worse at night, and especially when the disease is traceable to uterine derangement.

A weak continuous galvanic current is sometimes applied with advantage. A narrow terminal, connected with the positive pole, may be placed over or below each of the affected joints in turn; a sponge, connected with the negative pole, higher up the limb or nearer the spine.

Baths and douches (including even cold douches) are recommended by most writers; but Braun gives a warning against their use, except in a very gentle way. Tincture of iodine may be painted over the swollen articulations; or they may be strapped up with plaster; or the belladonna liniment may be applied to them; or the liniment. cantharidis, which Dr Garrod says is more convenient than an ordinary blister. Trousseau recommends that the affected parts should be buried in hot sand, at a temperature as high as can be borne, three times a day for an hour or two at a time. In certain cases stimulating applications, such as the cajeput oil, do good. As to the extent to which the patient should use the joints Dr Garrod suggests the rule that only such an amount of movement should be allowed as will not cause them to be more painful on the following day. The best climates are such as are warm, but dry and bracing.

[I venture to add confirmation of the author's recommendation of arsenic. Given in full doses, I have found it the most efficient drug in this disease, although no doubt cases occur in which it is useless. Patients have told me that as soon as their eyes begin to itch they know the pains and stiffness will be relieved. I have never seen any good from iodide of potassium and I think I have seen harm. Next to arsenic, steel and bark are the best drugs. But more important than either is cod-liver oil (first introduced by Dr Laycock for "rheumatism") with porter and a generous diet.

Of external remedies, I have sometimes found continuous galvanism the most valuable; in other cases it has failed. Hot douches, hot sand poured over the joints affected, hot baths, and blisters or other counter-irritation are all valuable. Warmth and flannel and powdered sulphur do good, cold and damp are harmful. Passive movement is necessary, and after the hot bath or douche the joints should be well shampooed. Moreover, the patient should be encouraged to persevere in movement, and to play the piano, or make any other exertion of the fingers.

Lastly, removal from Ireland, and if possible from England, to a warm, dry, and equable climate is invaluable. I believe most cases can thus be cured if the treatment is early adopted.—Ed.]

GONORRHŒAL SYNOVITIS

Distinction from rheumatism—Ætiology—Distribution—Symptoms—Sclerotitis—Prognosis—Treatment.

Sir Astley Cooper's 'Lectures on Surgery,' published in 1824, seem to contain the earliest notice of the fact that gonorrhœa may be followed by a painful disease of the joints.

This is commonly called *gonorrhœal rheumatism*, but Dr Pye-Smith, in a paper in the 'Guy's Hospital Reports' for 1874, has proposed to term it *gonorrhœal synovitis*, which name is, I think, to be preferred. Some of my surgical colleagues were until lately disposed to maintain that the relation between the two affections was merely accidental, but it is observed far too frequently to admit of such an explanation; Dr Pye-Smith collected twenty-nine cases from the medical records of the hospital in 1870-72; and during this time many others must have presented themselves in the surgical out-patient room. They all, without exception, occurred in men; Mr Brodhurst, however, in 'Reynolds' System' says that he has met with a few instances in women.* Senator, in Ziemssen's 'Cyclopædia' suggests that the toughness and thickness of the vaginal mucous membrane may afford an explanation of the rarity of gonorrhœal synovitis in the female sex. Nineteen of Dr Pye-Smith's patients were between twenty and thirty years of age; the youngest was eighteen, the oldest was forty-one.

According to Mr Brodhurst, the first affection is often directly preceded by exposure to cold and wet; he mentions the case of an officer serving in a tropical climate, who while suffering under gonorrhœa slept out of doors until after sunset, and woke in such great pain that he could with difficulty be removed to bed. It often happens that two and sometimes that three or more attacks of urethral inflammation in the same person are each followed by synovitis. Sir Benjamin Brodie recorded an instance in which, after two attacks from gonorrhœa, two later ones were attributed to irritation of the canal by the use of a bougie. In three of Dr Pye-Smith's cases a previous illness, which had been termed rheumatic fever, was said to have occurred; in five there was a history of rheumatism or gout in the patient's family. The length of time which elapses between the commencement of the gonorrhœa and the development of the synovitis is put by Mr Brodhurst at from ten days to three weeks; but Dr Pye-Smith found it to range from a week to nine months, and my own experience has been that it is often from six to twelve weeks. Sometimes the urethral discharge continues to be profuse after the appearance of the joint-affection, but in most instances there remains only a slight gleet. Indeed, the patient is very apt to omit all mention of its presence, or even to deny that he has had any venereal complaint. Careful inquiry must therefore be made as to the previous existence of gonorrhœa in all cases of supposed rheumatism occurring in young male patients, especially when the more characteristic phenomena of acute rheumatism are wanting; and it is often right to insist upon an inspection of the urethral meatus. However, even when a discharge is found, we may sometimes still be in doubt as to the real nature of the joint-affection if the patient have had previous rheumatic attacks, as was the case with a man recently under my care. And another point which must not be forgotten in diagnosis is that gonorrhœa is sometimes followed by fatal pyæmia, with suppurative inflammation of the joints (p. 551).

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* Mr Davies Colley, in the 'Guy's Reports' for 1882, and Dr Church, have observed the same.

Locality.—Writers generally say that the knee is the most frequent seat of gonorrhœal synovitis. Senator mentions that among five cases which came under his observation the left knee alone was affected in four, both knees in the remaining one. But at Guy's Hospital it has long been taught that the feet are the parts most apt to suffer, and this is borne out by Dr Pye-Smith's analysis of his twenty-nine cases, for in more than twenty of them the ankle, sole, heel, or instep was attacked, while the knee was affected in only fourteen, the wrist in six, the shoulder in three, the hip and the elbow in one each. The toes often were painful, but not the fingers. The pain was usually worse at night, and was always described as of a dull, constant aching character. There was in most cases moderate œdema, and occasionally a slight inflammatory blush as well as local heat. The knee was the only joint in which marked effusion was detected.

Symptoms.—The pyrexia in gonorrhœal synovitis is almost always moderate, and I believe that in many instances the temperature of the body remains normal. Hyperpyrexia is unknown. The disease seems to have no tendency to affect the heart. Dr Pye-Smith mentions that in one patient, a man aged twenty-four, there was a systolic basic bruit, but he suggests that this was "functional."

It is, however, frequently accompanied by ophthalmia. Thus, what first drew Sir Astley Cooper's attention to the subject seems to have been the case of an American gentleman, who came on account of a gonorrhœa, and said that two previous attacks had each led to inflammation of the eyes, and a few days later to swelling of the joints. Precisely the same sequence occurred on the third occasion, under the observation of Sir Astley himself. Dr Pye-Smith found that this eye-affection developed itself in seven of the twenty-nine cases which were collected by him. It usually attacked first one eye and then the other. It was commonly attended with injection of the fine radiating vessels of the sclerotic which surround the cornea, and sometimes with iritis. It always subsided in a few days under treatment by cold bathing, covering from light, and applying atropine drops. In more than one instance it returned, after having disappeared.

Events.—Gonorrhœal synovitis commonly runs a tedious and protracted course, lasting for several weeks, and even for months, notwithstanding treatment. In hospital practice I can look back upon several instances in which the obstinacy of a supposed "subacute rheumatism" led to the discovery of the fact that the patients had gleet, the presence of which had been concealed by these patients. Recovery seems to have occurred in all the cases which were collected by Dr Pye-Smith, although some of the patients left the wards before they had entirely lost the pains and stiffness of their joints. But Mr Brodhurst mentions one instance in which the hips, the knees, and the jaw were all ancylosed, and another in which in the course of five years the whole skeleton became fixed, including even the articulations of the vertebræ and those between the atlas and the occiput, so that the head itself could not be moved. That patient had had several attacks of gonorrhœa, each followed by "rheumatism." About six months after the last attack, and when he was only just able to walk about, he was unfortunate enough to espouse a woman who had an occluded vagina, so that painful attempts to consummate the marriage were altogether unsuccessful. Very soon afterwards, although there was no urethral discharge, the articular inflammation recurred; and on this occasion it led to the terrible results already described. Mr Brodhurst also says that ancylosis has occurred in all the female patients in whom he has seen gonorrhœal synovitis.*

Treatment.—The best medicine in this disease seems to be iodide of potassium, which may be given in full doses, up to thirty or forty grains daily. The patient must be kept in bed and upon light diet, at least during the first few weeks. Mr Brodhurst insists upon the importance of placing the affected joints upon splints. He says that leeches sometimes do harm rather than good. He has seen the Turkish bath very useful, and instances a gentleman who was lodged in a house attached to one of these baths, so that he could be carried down into the hot chamber every day; when profuse perspiration was obtained the pain, which was very acute, left him —but, I suppose, only for the time.

In chronic cases blisters may be applied with advantage, or liniment of iodine, or mercurial ointment. When one or more of the joints has become fixed, it is often advisable to give chloroform, and to break down the adhesions by force. Mr Brodhurst says that in this way it is possible in many cases to restore perfect mobility, even when ancylosis had appeared to be complete.

[CHARCOT'S JOINT-DISEASE.—*Arthropathie ataxique.*—It is to the acumen of Professor Charcot (1868) that we owe the recognition of a very curious chronic affection of the joints which is liable to occur in the course of Tabes dorsalis (locomotor ataxia). It is insidious in its origin, extremely chronic in its course, and remarkably wanting in all local signs of inflammation.

The joint affected, most often the knee, gradually becomes completely disorganised. The synovial membrane and cartilage disappear, and the articular ends of the bone undergo singular atrophy, by which the tuberosities of the tibia, the condyles of the femur, or (if the hip is affected) its entire head and neck, are absorbed. With this there is little or no hypertrophy. At last the ligaments are so relaxed and the ends of the bones so altered that the joint swings in all directions like a flail.

Whether this affection is a variety of Osteo-arthritis deformans, and what are its relations to gonorrhœal arthritis and even to syphilis, are points which have been much disputed.

Charcot's "chronic atrophic arthritis," as we may call it, was briefly alluded to in the account of locomotor ataxia in the first volume of this work (p. 459). A discussion of its nature by Sir James Paget, Mr Hulke, Mr Hutchinson, Dr Duckworth, Mr Lucas, and other pathologists, in which many cases were recounted and specimens shown, will be found in the eighteenth volume of the 'Clinical Society's Transactions,' and an earlier paper by Dr Buzzard ('Path. Trans.,' vol. xxxi, p. 193, 202) should also be consulted. Two good cases were published by Dr Charles Atkin in the Manchester 'Medical Chronicle,' April, 1885, and six by Dr Sydney Roberts in the Philadelphia 'Medical News,' February 14th, 1885.—ED.]

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* [Though peculiarly tedious and obstinate, gonorrhœal synovitis when once cured does not return—another difference from rheumatism. Numerous instances, however, prove that a fresh urethritis will produce a fresh synovitis, and in this way the same patient may suffer twice, thrice, or even oftener from "gonorrhœal rheumatism."

In my experience neither colchicum, salicin, nor iodide of potassium are of any use in this affection. Blisters, with good feeding, cod-liver oil, and iron or bark, are the best treatment. See an excellent paper by Dr Thomas Bond (published before my paper in the 'Guy's Reports') in the 'Lancet,' March 23rd, 1872.—ED.]

DISEASES OF THE BONES

RICKETS

History—Anatomy of rachitic limbs—Thorax—Pelvis—Skull—Histology—Course—Event—Symptoms—Ætiology: Age, sex, climate, food—Relation to tubercle—Pathology—Diagnosis—Cranio-tabes—Treatment, preventive and curative.

About two centuries and a half ago certain English physicians drew the attention of the profession to a disease affecting the bones of children, which they supposed to have recently sprung up in England, where it was first observed in the counties of Devon and Somerset. It had already become known by the popular appellation of *the Rickets,* a derivative either of a Dorsetshire verb "to rucket" (= to breathe laboriously) or of "rick" (= elevation or lump), or again, according to Trousseau, of a Norman word "riquets" applied to deformed persons. The term *Rachitis* (or Rhachitis) was first proposed for it by the famous anatomist and physician, Glisson, of Cambridge, who, I think, really chose that name on account of its similarity in sound to the other, although in his work on this disease (1650) he offers to his readers the choice of a Greek root (*ῥάχις*), on the ground that the dorsal spine is one of the first parts to be attacked. In a thesis by Whistler (said to have been published at Leyden in 1645) the complaint was spoken of as *morbus puerilis Anglorum,* and probably this led foreign writers to call it *morbus Anglicus.* It was even supposed to have spread from England to the Continent. But there is little doubt that it had really existed among children from time immemorial on both sides of the Channel. It is true that only a few allusions to the disease are to be found in older works, but according to Senator there is an antique statue of Æsop which exhibits deformities characteristic of it.*

Rickets may be defined as a defective and perverted development of the osseous tissue of growing bones, attended with an enlargement of certain parts of them, and leading to a distortion of their shape. In its extreme forms it affects the whole skeleton of the child, but it often begins in some particular region, and it may even remain limited to the chest, or to the head, or to some of the limbs, at least so far as its more obvious manifestations are concerned.

Anatomy.—In the arms and legs the earliest sign of the disease is an increase in size of the ends of the long bones. This is particularly marked at the wrist; the radius and ulna form a flattened pear-shaped prominence overhanging the small hand. Something similar may be seen in the case of the ankle. The joint of the knee appears as a hollow or depression between the projections of the articular extremities of the femur and of the tibia, and hence the expression "doubling of the joints," which has sometimes been used as synonymous with rickets. If the affection advances, the limbs become curved. In the forearm, the bones almost always bend so that they are convex towards the extensor surface; in the upper arm the character of the distortion is less uniform. In the thigh the rule is that the femur is arched with its convexity looking forwards and outwards. The knees may thus be thrown far apart, and the patient becomes "bow-legged." The shape assumed by the tibia and fibula varies in different cases. They often carry downwards and inwards the curve formed by the femur on each side, so that the ankles meet one another, although the knees do not. But in other instances they are themselves bent so as to be convex outwards or inwards; in the latter case the feet are widely separated, and sometimes each leg presents a rather sharp angle, projecting forwards at the junction of the middle and the lower thirds.

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* [A cast of this statue has been lately added to the collection of antique casts in the South Kensington Museum.—Ed.]

The chief cause of these various deformities appears to be the yielding of the bones to the traction and pressure to which they are subjected. They are, in fact, so soft that very little force is required to bend them, as can easily be demonstrated after death. Thus the curves in the forearms and upper arms are probably due to efforts made by the child to raise itself by laying hold of fixed objects with its hands, and to other like movements; there is often a very marked angle at the insertion of the deltoid into the humerus. The more common distortions of the bones of the thighs and legs seem to be caused by the weight of the body in the erect posture, but the angular bend which is found just above the ankles is, I think, due to pressure transmitted to the tibiæ from the insteps and feet in crawling about upon the floor, a favourite mode of progression among such little patients. It has, indeed, been objected that in stillborn fœtuses, believed to be rhachitic, similar changes have been observed in the shape of the limbs, but the objection seems to be of little weight, for surely they may, while within the uterus, undergo compression powerful enough to deform them.

Another effect of the softening of the bones is that a very slight accident suffices to partially break them. Such "green-stick" fractures, as they are called, may be caused by abrupt movements; sometimes several of them are seen in the same child. Their effects, of course, complicate and alter in various ways the more regular distortions resulting from the disease, and all the more because, interfering but little with the movements of the affected part, and giving rise to no marked increase of pain, they are very apt to escape notice until a large quantity of callus has been thrown out.

Of far more importance as regards the patient's health, though perhaps less conspicuous to the eye of an untrained observer, are the changes produced by rickets in the shape of the *chest*. Here, again, the first indication of the disease is an enlargement of the growing ends of the bones; namely, of the ribs just where they join their cartilages. The consequence is the formation of a series of little nodules, which can be easily felt and may even be seen through the integuments, and which are arranged in a vertical line, slanting outwards as it passes downwards on each side of the sternum. This "beading" of the ribs, as it is termed, is sometimes the only discoverable sign of rickets which a child may present; it must therefore be carefully looked for whenever the existence of this disease is suspected. But, further, there is in most cases a more or less considerable alteration in the form of the thorax itself, by which its capacity may be greatly reduced. If one watches a healthy child who is suffering from extreme dyspnœa dependent on obstruction of the larynx or trachea, one may observe that at each inspiration the middle parts of the ribs are forcibly dragged inwards. This is especially the case with those ribs which lie towards the base of the chest on each side, but not where any solid organ, such as the heart or the liver, lies behind them. The cause of it is that they are unable to resist the atmospheric pressure when they are no longer supported by the counter-pressure of air entering the lungs freely from the mouth. Now, in rickets, it would seem that the mere elasticity of the lungs is sufficient to turn the scale and to prevent the lateral portions of the softened ribs from moving outwards when the child draws its breath; or it may be that this state of things is simply brought about by very trifling and transitory affections of the bronchial tubes; although I do not know that it has been proved to occur in those who have never had even the slightest cough or catarrh. In either case, the effect is not transitory, as it would be under normal circumstances; but there arises a persistent flattening, or even a depression, of the chest walls. This generally runs, as a vertical broad shallow groove, downwards and outwards from just below the fold of the axilla on each side; as it approaches the margins of the costal cartilages it forms an angle and slopes away to each side, now lying almost parallel with the diaphragm. Or one may describe two sulci—the one nearly perpendicular, the other horizontal—meeting at an obtuse angle a little below the base of the xiphoid cartilage. The vertical groove is generally said to be formed by the ribs themselves, outside their cartilages, and outside their beaded ends. But Dr Gee ('St Bart. Hosp. Rep.,' vol. iv) has pointed out that the beads sometimes occupy the bottom of the groove, and that in exceptional cases they may lie to the outer side of it, so that, in fact, it corresponds with the cartilages only and not at all with the bones. One result of this depression of the ribs is that the higher abdominal viscera are pushed out from below the ribs; the liver projects beyond the costal margins more than in a healthy child; and, as the intestines are commonly very full of gas, the belly becomes protuberant and contrasts strongly with the narrow chest. Another effect, according to Sir William Jenner, is the production of a white friction-patch on the surface of the heart, just above the apex of the left ventricle, where the fifth rib presses on it (see his well-known Lectures in the 'Medical Times' for 1860, vol. i). And yet another is an increase in the antero-posterior diameter of the thorax itself; the sternum becomes, as it were, inflated, and the dorsal vertebræ form a rounded curve.

These changes together constitute what is commonly called the *pigeon-breast.* They are almost always associated with the presence of emphysema in the anterior edges of the lungs, beneath the projecting sternum; while, in correspondence with the flattened ribs, one may often notice a collapsed condition of the inferior edges of the lungs, and even of parts of their lateral surfaces. In contrast with the dorsal, the cervical and the lumbar vertebræ have their natural forward curves exaggerated in rickets. Another feature of the disease is that the clavicles are much more bent than in the normal state, and carry the shoulders further backwards, with the effect of increasing the apparent prominence and narrowness of the upper part of the chest.

In the *pelvis* various deformities occur, but these are not obvious during the existence of the disease, and are only important because in females they may permanently narrow the cavity and obstruct parturition. In most cases the brim appears to assume an hour-glass or oval shape, the pubes being approximated to the sacrum, but it may be triangular or rostrate.

The growth of the body generally is retarded in rickets; a child two years of age may be taken for not more than six months old; a boy of twelve may be no taller than he ought to have been at three. Among forty-two cases in which Ritter von Rittershain (1863) made careful measurements at ages between four months and three years, there was only one in which the length of the body was not from one and a quarter to two and a half inches below the mean length in healthy children at the same ages.

Rickety infants, however, are not infrequently fat, and sometimes to an excessive degree.

The face is peculiarly backward in its development: the jaws remain narrow, and dentition is late and irregular. It is not uncommon for a rachitic infant a year old to have cut none of its teeth; and when two or more of the incisors have appeared before the commencement of the disease, they are sometimes without successors for several months. The teeth themselves are imperfectly formed; their enamel is defective; in a year or two they turn black and break off, or fall out. Dr Gee has pointed out that the second dentition is also delayed.

In marked contrast with all other parts of the skeleton is the appearance of the *skull.* This is disproportionately large, and so much so that until recently it was generally believed to be actually larger than in healthy children of the same age. Ritter von Rittershain has, however, shown by accurate comparative measurements that the enlargement is generally only apparent.

With regard to the state of the brain I find some discrepancies of statement among different writers. Trousseau maintains that the softness of the cranium allows of the more easy development of the nervous centres, and so accounts for the possession by cachectic children of intellectual faculties in advance of their age—a fact, if it be a fact, which it would be more reasonable to attribute to the habits induced by their unfitness for muscular exertion and association with adults. Dr Gee thinks that the growth of the brain is really dwarfed, like the rest of the body, and that fluid is commonly effused into the ventricles to fill up the empty space within the skull. But it seems to me most unlikely that the cranial cavity would be large, were it not to hold a large brain; and it is certain that in many cases in which the head appears to be increased in size there is no excess of fluid. On the other hand everyone knows that, as a complication of rickets, hydrocephalus is of frequent occurrence, and the affection known as hypertrophy of the cerebral substance is sometimes met with (vol. i, p. 555).

The form of the cranium also is altered. It has been described as being elongated; but in reality it is much more often square-shaped, and flattened on the summit, in consequence of the fact that the fontanelles fail to close at the proper time; clinically, indeed, this is perhaps the most important of all the symptoms of the disease, except the beading of the ribs. The principal fontanelle, certainly with more or less irregularity of form, not uncommonly remains open up to the age of three years, or even longer. Moreover, there is often separation of the bones, where they meet to form sutures. Their margins, being the growing parts, are generally more or less thickened; sometimes one can feel a distinct ridge along the vertex and even down the front of the forehead. On the other hand, there is often an irregular thinning of the occipital bone—a condition first described by Elsässer, and commonly known by the name which he gave to it of *cranio-tabes* (cf. vol. i, p. 136). The way to detect it is to grasp the head with the two hands, and to make very gentle but firm pressure with the tips of the forefingers over all parts of the surface of the bone in succession. One may then find that certain small spots, generally near the lambdoidal suture, yield and become indented, just as though the osseous tissue were replaced by a piece of cardboard.

Histology.—The microscopical changes in rickets are very interesting and have been carefully studied within the last few years. If with a strong knife one cuts through a rib and its cartilages, across the plane of the union between them—or if one divides the end of a long bone, so as to expose on the face of the incision the junction between its shaft and one of its epiphyses—certain deviations from the normal appearances are at once obvious, even to the naked eye. The so-called "zone of proliferation" of the cartilage ought to be a well-defined, straight, narrow, bluish-white seam, perhaps one sixteenth of an inch in thickness; and the yellow "ossifying zone" beneath it ought to be still narrower. Instead of this, however, the "zone of proliferation" is considerably thickened, reddened, and of a soft spongy texture. Moreover, the meeting line between them is most irregular and sinuous, with promontories and islands of bone, and even medullary spaces, projecting far into the cartilage. Rindfleisch aptly sums up these changes by saying that the processes which prepare the way for the conversion of cartilage into bone are morbidly accelerated, without the actual ossification keeping pace with them. So, again, beneath the periosteum. Here, instead of an almost inappreciable quantity of embryonic tissue, there is in rickets a soft, red, vascular layer, perhaps one twelfth of an inch thick, which has been compared with the pulpy substance of the spleen. It sends processes into the superficial vascular canals, and often has embedded in it numerous minute osseous granules or trabeculæ, which tear away with it from the shaft, leaving the latter rough. The whole of the interior of the bone also, including the medullary cavity, is unnaturally red and vascular.

In thin sections, and with the aid of a microscope, the exact nature of the affection can be traced more minutely. The broad, bluish-white zone contains long columns of proliferated cartilage-cells, thirty or forty deep. And, unlike what occurs in the normal process of ossification, these cells can easily be seen to be directly transformed into stellate bone-cells, each of which, however, remains surrounded by a delicate ring, corresponding with the former cartilage capsule. Rindfleisch says that the homogeneous chalky appearance of this "cartilage bone" enables it to be recognised with the naked eye, even when it is embedded in regular osseous tissue.

The chemical constitution of the bones in rickets has been several times investigated, and the proportion of inorganic to organic matter has been found greatly below what is normal. The analysis of Friedleben, however, published in 1860, made the percentage of earthy salts from 33 to 52, which is considerably higher than that given by earlier inquirers, although still much less than the percentage of 63 to 65 obtained from the bones of healthy children.

Course.—Rickets generally runs a somewhat chronic course, but subsides, under favourable circumstances, at the end of a year or two. Some writers, however, have described an acute form of the disease. Senator records the case of a child, four months old, who became feverish, and in whom the epiphyses of several of the long bones of the limbs were swollen and very tender, but without redness; the affection subsided entirely in about six weeks. Feist is said to have been the first to observe instances of this kind; but I find, on referring to them, that his were simply cases of rapidly fatal multiple abscesses of joints occurring in very young infants. It does not appear that any example of "acute rickets" has hitherto been identified by its histological characters.*

Event.—When recovery from the disease takes place, the bones lose their soft spongy appearance, and become actually denser and harder than natural. The articular ends are no longer enlarged, but perhaps it is not due to their absorption, but rather to their being overtaken in their growth by other parts, that the normal proportions are restored. Many of the deformities which are so conspicuous in young children seem slowly to disappear, at least when they do not exceed certain ill-defined limits. It used, however, to be traditionally taught at the Hospital for Children in the Waterloo Road that although tibiæ which were laterally curved might become straight in the course of time, a similar change never occurred when they were sharply bent with the convexity forwards near the ankles, in the manner which, I believe, is attributable to crawling on the floor. The pigeon-breast is very generally permanent; and in too many cases the limbs, as well as the trunk, remain horribly distorted for the rest of life. Even when there are no very striking alterations in the shape of the bones, one can often recognise the fact that a person was rickety in childhood by his short stature, by his square thick-set frame, and by his large protuberant head. Such persons are often erroneously supposed to have suffered from hydrocephalus.

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* [Lately, however, a remarkable form of what may be called "acute rickets associated with purpura" (or scurvy ?) has been described by several authors: Dr Cheadle ('Lancet,' 1878), Dr Gee (who called it "osteal or periosteal cachexia," 'St Bart.'s Hosp. Rep.,' vol. xvii), Mr Thomas Smith ('Path. Trans.,' vol. xxvii), Dr Goodhart ('Dis. of Children,' p. 556), and Dr Barlow, whose excellent account of eleven cases with two autopsies will be found in the 'Med.-Chir. Trans.,' vol. lxvi, p. 159.—Ed.]

Most writers express doubts as to whether death is ever caused by rickets alone, apart from any complications. Dr Eustace Smith, however, says that he has seen it directly fatal, with extreme dyspnœa and lividity. As a rule, if the child succumbs, it is to diarrhœa, or bronchitis, or laryngismus stridulus; or perhaps to croup or pneumonia, or to one of the exanthemata.

Following Sir William Jenner, some English observers attach considerable importance to a change in the liver and spleen and lymphatic glands, which he described as an "albuminoid infiltration." Dr Dickinson has investigated the microscopical characters of this affection, which he finds to be an overgrowth of the fibrous tissue in the portal canals of the liver and in the trabeculæ of the spleen respectively, with some excess of cellular elements also. The organs, he says, feel hard, dense, and elastic; the liver shows yellowish acini, each surrounded by a thin pinkish or grey line; the spleen, which may be so large as to extend below the umbilicus, is of a deep red or purple colour, besprinkled with smooth, white spots, or mottled into a pale buff. The glands are moderately increased in size, tough, white, and opaque. Dr Gee, however, states that in the majority of rickety children who die with an enlarged spleen, its appearance differs in no respect from that of the spleen of ague, or of inherited syphilis, or of cachexia due to unknown causes. He thinks that the affection is really a result not of the rickets, but of the general state of ill-health which caused the rickets; and I am very much disposed to agree with him. Everyone admits that, as a rule, it subsides under treatment. Dr Dickinson, like Sir William Jenner, connects it with the occurrence of emaciation and anæmia, but he does not speak of it as being in itself ever the cause of death.

General symptoms.—Hitherto I have made no allusion to any other symptoms of rickets except such as are afforded directly by the perverted development of the bones and teeth. Writers, however, describe it as being ushered in by prodromata, consisting perhaps of sickness, diarrhœa, and tumefaction of the abdomen, with languor, drowsiness, loss of appetite, and febrile disturbance. We shall presently see that the disease is essentially the result of enfeeblement of the general health; and it appears to me that all of those supposed early signs of rickets ought to be regarded either as other and independent effects of the same cause, or as being themselves accessory causes. At any rate, it is a point of great practical importance to remember when one is called to a case of croup or of bronchopneumonia that one often has to deal at the same time with an advanced state of rickets, although the mother may have thought the child in fairly good health up to the beginning of its acute illness. There are, indeed, certain reasons which make it difficult for us to look upon rickets as a mere affection of the growing bones and of structures allied to them, although I do not think that they compel us to throw it into the vague class of "general diseases."

These reasons are connected with some minor symptoms, which often lend considerable aid in diagnosis, and which are very curious, although as yet they are altogether unexplained. One is a peculiar *restlessness* at night, which causes the child, even in cold weather, to kick off the bedclothes, or to throw its naked legs out upon the counterpane, as often as it is covered over by the nurse. Another is a tendency for profuse *perspiration* to break out upon the head, and the neck, and the upper part of the chest, especially during sleep. Elsässer laid much stress on this in connection with his *craniotabes* (1843). Dr Gee has shown that the disease *per se* causes no elevation of *temperature* even in the evening. A third sign of rickets is a *sensitiveness* of the body and limbs, so that the child lies motionless, and dislikes being touched, or moved, or handled; while, if lifted by the armpits, and tossed up and down, it at once begins to cry. The tenderness appears to be partly in the bones and periosteum, but Jenner and Dr Gee have pointed out that gentle pressure upon the muscles of the loins or abdomen is sometimes no less painful. Indeed, the muscles are commonly soft and flabby, and more or less wasted. In severe cases the child is almost always unable to walk, or even to stand, even though he may have been on his feet for some time before he became rickety. Dr Gee speaks of a "pseudo-paraplegia" under such circumstances. Jenner relates the case of a girl, six years old, who could neither change her position in bed without assistance nor lift her arm an inch from the surface on which it lay; even at a later period, when she had greatly improved, she was obliged to be tied into a chair with a pillow at its back to support her head; and if the head fell forward the nurse had to raise it for her. She afterwards recovered so as to walk without assistance.

Age.—That rickets is generally a disease of early childhood is admitted by all observers, but they are not agreed as to the exact limits of the period within which it is most apt to arise. The common statement is that it is from the sixth month to the end of the second year; corresponding, in fact, with the first dentition. And when one inquires closely into cases which are said to have begun later than this one usually finds grounds for suspecting that a slight form of the affection had existed for some time previously, although it may recently have undergone a more rapid increase. Dr Gee, who collected 635 cases, is even disposed to agree with von Rittershain in thinking that in reality its commencement does not often date after the end of the first year. The latest case that he had himself observed was one which seemed to have begun at twenty months. The child, who showed considerable beading of the ribs, had cut the first tooth at six months, and at twelve months it had been weaned and had walked; six weeks before it came under Dr Gee's notice it had begun to get weak in the legs and loins, and during the last three weeks it had sweated much. Even in that instance the absence of the disease at an earlier period was merely a matter of inference. And there can be no doubt that the statistical tables which have been drawn up by different writers are largely open to the same objection, so that in fact very little value can be attached to them. Some have supposed that the disease may develop itself in young adults, but this idea appears to have been based upon erroneous views as to the nature of curvature of the spine or of certain joint-affections. Indeed, it seems scarcely possible that an identical morbid process should arise when the process of ossification has in the main been completed; and in the future no such case would be accepted without full details as to its histology. On the other hand, it is certain that rickets may be present before the sixth month. Dr Gee speaks of unquestionable beading of the ribs in infants only three or four weeks old. In such cases it seems probable enough that the starting-point of the disease was in intra-uterine life. Whether it can ever be recognised at the time of birth is still doubtful. A few supposed instances of such an occurrence in stillborn fœtuses have been recorded,* but Urtel (1873) and Eberth (1878) have each found that the histology of the affection described under that name is altogether different, the process by which the epiphysial cartilages normally undergo conversion into the bone being arrested at a much earlier period, and before the cartilage-cells have begun to proliferate and to arrange themselves in vertical columns. The most conspicuous character of such cases is the extremely stunted form of the limbs.

Sex.—With regard to the relative liability of boys and girls to the disease, writers have made opposite statements; the only conclusion seems to be that it is equally common in the two sexes. Its absolute frequency probably varies in different countries, being greater where the climate is damp and cold. The statistical results hitherto collected are not exactly comparable with one another; it will be sufficient to say that Dr Gee found rickets in no less than 30·3 per cent. of all children under two years old brought to him at the hospital in Great Ormond Street in 1867. It is believed to be far less common among those who live in the country than among the inhabitants of crowded cities, where children are apt to get very little light or air.

Ætiology.—Many attempts have been made to find a definite exciting cause for the disease; one observer endeavoured to trace it to too prolonged lactation, another to premature weaning. Guérin is generally said to have given principal support to the latter view by some experiments on young puppies, whom he deprived of their mother's milk, and fed with meat; the more recent investigations of Tripier, however, have shown that although animals treated in this way become sickly and die, they are not really affected with rickets. Another suggestion has been that it is due to a deficiency of lime and of phosphoric acid in the food. Chossat and the younger Milne Edwards succeeded in producing curvatures of the bones in pigeons and dogs by cutting off the supply of their nutritive salts; but Friedleben has since found that even where atrophy of the osseous tissue is thus induced, the changes characteristic of rickets are wanting. In fact, clinical observation itself points strongly to the conclusion that although defective or improper food is a very frequent and important cause of rickets, it acts indirectly, and in all probability by producing a generally enfeebled state, and the same may be said of other conditions to which the disease has been attributed, such as syphilis in the parents, phthisis, emaciation, exhaustion, anæmia—and even old age. Sir William Jenner says that it is very doubtful whether impairment of a father's health has any influence in inducing rickets in his children, whereas von Rittershain thought that he traced the disease to the presence of some chronic tuberculous disease in the father more often than in the mother. But the truth is that among the poor it is impossible to isolate causes of this kind; a husband's illness may deprive the wife of nourishment, throw much heavy work upon her, and in many different ways render her likely to bear weakly infants. So, again, even when the parents of a rickety child are, one or both of them, rickety, it seems doubtful whether the disease is really transmitted; perhaps, after all, the state of their health acts only like any other common debilitating influence. A point of great importance, on which Sir William Jenner has laid stress, is that the first child of a family, or even the first two or three, may be found free from rickets, where later ones are affected by it; and, again, that if once a woman has borne a rickety infant, those that follow are almost sure to become victims of the disease. This is due not only to the progressive enfeeblement of the mother's health by repeated child-bearing, but also, among the poor, to the overcrowding of space and deficiency of clothing and food which are implied by a large family; and perhaps among the middle classes, to the way in which children are sometimes kept indoors, when there is but one nursemaid for several of them.

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* [By Jules Guérin, 'Mem. sur les caractères de Rachitisme,' as early as 1839. See cases of so-called fœtal rickets by Dr Thomas Barlow and Mr Shattock, 'Path. Tr.,' 1881, pp. 364, 369.—Ed.]

The relation of rickets to *tuberculosis* seem to me to require further investigation at the hands of pathologists untrammelled by previously formed opinions. Having regard to the similarity of the conditions that favour their development, one would certainly have expected that they would often have been found in the same children. And Sir William Jenner, although he contrasts the two "diatheses," adds that rickets does not by any means exclude tubercle. Dr Eustace Smith, however, says that rickets never occur in children in whom the tubercular "disposition" is well marked; but then this statement is a matter of course, because the features supposed to indicate that disposition are of themselves a proof of the absence of rickets; and we come at once back to the question discussed at p. 975 of the first volume of this work, whether there is really a single class of individuals who can be said to be specially tubercular. I have already quoted the statements of Ritter von Rittershain as to the inheritance of rickets from tuberculous fathers, but Sir William Jenner refers to a table made for him by Dr Edwards which appeared to show that phthisical parents are actually less likely than non-phthisical parents to have rickety children.*

It was long ago suggested that the immediate cause of the changes in the bones in rickets was the action of lactic acid, dissolving out the lime salts from their substance. The acid was said to have been detected not only in the bones themselves, but in the urine; it was supposed to be formed in excessive quantity in the alimentary canal from milk and other articles of food. Some chemists also stated that more than the normal amount of phosphate of lime was excreted by the kidneys. The modern investigation as to the histology of the disease would obviously have rendered such a theory untenable, even if it had otherwise had a chance of acceptance. Recently, however, another view has been promulgated, in which the acid plays a different part. In 1871 Dr Wegner, of Berlin, in the course of some experiments upon young animals with minute doses of phosphorus, found that if, while administering the poison, he withheld lime salts from the food, there arose an affection of the bones precisely like rickets, as it is seen in the human subject. He supposed that the phosphorus was a stimulant to the osseous tissue. Now, Heitzmann has since stated that lactic acid is capable of acting in the same way. The hypothesis, therefore, as given by Senator, is that the disease is the combined result of the irritant influence of that acid upon the growing bones, and of a deficiency of phosphate of lime, consequent either on their being too little of it in the food, or on its being carried away through the bowels by diarrhœa. But at present this theory seems to rest upon too slender a foundation of facts.

The *diagnosis* of rickets is very easy when it is fully developed. As a source of fallacy in regard to the *cranio-tabes* of Elsässer, I may perhaps mention a case of cerebellar tumour (referred to in vol. i, p. 532) in which a somewhat similar thinning of the occipital bone was observed. The only real difficulty is as regards early cases in young children. I must confess that I have sometimes been in doubt as to what constitutes "beading" of the ribs, as distinguished from the slight roundness of their ends which is normal.

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* [The relation of rickets and particularly of craniotabes to syphilis was discussed at the International Medical Congress of 1881 ('Trans.,' vol. iv, p. 35) by MM. Parrot, Guérin, and Bouchut, of Paris, Dr Rehn, of Frankfort, and other pathologists. See also on this point an admirable paper, with tables, by Drs Lees and Barlow in the 'Path. Trans.,' vol. xxxii, p. 323.—Ed.]

The *prognosis* of the affection, if left unmodified by treatment, is said to be more grave in proportion as the child is younger at the time of its commencement. It is therefore extremely important to be on the look-out for it whenever an infant at the time of the first dentition begins to fail in health, or suffers from any trifling disorder, such as relaxation of the bowels. For, as will have been perceived from what has been stated in regard to its ætiology, rickets is eminently a disease that can be prevented; it is also one that can be cured.

Treatment.—The chief thing of all is to attend to the food. When an infant is suckled, the breast should during the first six weeks be given every two hours, except from eleven p.m. to five a.m., during which interval the mother or the wet nurse should be allowed to sleep; at a later period every three hours, or still less frequently. It is very wrong to let a child lie asleep with the nipple in its mouth, although nothing is more common than for it to be kept at the breast all the night while the mother herself is in a sound slumber. If a baby does not thrive, one cause for it may be that the milk is insufficient in quantity or too poor. According to Dr Eustace Smith it is a sign that this is the case when the infant falls asleep while sucking; or one may notice that it sucks away at its thumbs until they become quite raw. If something in addition to the breast milk is required, one may employ cows' milk or asses' milk, sweetened a little, and perhaps diluted slightly with water according to the age. Neither buiscuit powder, nor any other farinaceous food should be administered to very young infants. The secretion of saliva (there being no physiological need for it) appears not to be established, under normal conditions, before the third month; and it is believed that up to that time all starchy matters pass through the intestine unaltered, and are discharged with the fæces. But I must confess that I have seen some well-grown children whose parents had been in ignorance of this rule, and had brought them up in direct opposition to it. Liebig's malted food, however, may safely be used, mixed with milk, even a week or two after birth. I am told, indeed, that some infants will not take it; and there is no doubt that what suits one baby perfectly may not do at all for another. Swiss milk, and the other concentrated preparations of milk, are often given to young children; but I agree with those who hold that they are not to be recommended. Swiss milk undoubtedly fattens rapidly; but the more important tissues seem not to be equally well sustained by it, and infants brought up upon it are very apt to succumb if attacked by diarrhœa or by other ailments. Probably these effects are attributable to the quantity of sugar contained in it; but I must confess to a prejudice against the attempt to preserve artificially a substance so liable to decomposition as milk. And the same feeling would lead me to hesitate about employing Nestle's compound, although it seems to have met with wide acceptance.

After six months it is always advisable that the mother's milk should be supplemented either by one of the farinaceous foods or by Liebig's maltine. At eight months, a little mutton-, or chicken-broth, or beef tea, may be given with advantage. At ten or twelve months the child should be weaned.

When a feeding-bottle is used, the most extreme care is required to keep it and the tube connected with it clean, so that it may not turn the milk which is put into it sour. It should be scalded out every time it is employed; and the tube and nib should be always kept in water.

No one who had not witnessed it would believe how utterly wrong is in most cases the management of young children belonging to the lower classes. At a very early age they are allowed to have bacon, fried fish, potatoes, and beer. If brought up by hand, they perhaps receive cornflour, or some other substance which is little better than pure starch; and it is often made up with water, instead of with milk. If suckled, they are not weaned until they are eighteen months or two years old; but, long before this, they feed with their parents at meals, and eat exactly the same things. A piece of bread and butter is constantly in their hands, to keep them quiet.

Even at a comparatively advanced age, a child affected with rickets requires certain precautions to be taken, which are not equally necessary for those who are healthy. Not only must its food be nutritious and digestible; it must also be easy of mastication, if the teeth are few in number or decayed. Thus one often has to direct that all the meat should be finely powdered in a mortar, and that the potatoes should be mashed, and all the lumps carefully picked out.

When indoors it should be kept lying down, and should not be encouraged to attempt to walk, so long as the bones are soft. I have even used splints projecting below the feet for the purpose of rendering such attempts altogether impossible. So far as I have been able to learn, mechanical appliances are of very little service in straightening the spine or the limbs. Sir William Jenner, however, has found, where the ribs were inclined to yield, that a well-adjusted bandage round the abdomen was useful by retarding the descent of the diaphragm.

At night the child should sleep on a hair mattress, and if its head is tender and inclined to perspire it should have a horsehair pillow made with a hole in the centre so as to remove all pressure from the occiput. Dr West has seen this give quiet sleep for the first time for weeks. A thin linen nightcap may also be worn, which can be changed two or three times if necessary.

Bathing is of considerable importance. The child should be sponged with warm soap and water once or twice a day, or, according to the season, with tepid or even cold water, in which sea-salt may be dissolved. Dr West recommends tan baths. They are made by adding to the water a decoction of oak bark. The formula for this is to take three handfuls of the bruised bark, and to boil it in a linen bag in three quarts of water for half an hour.

Drugs.—Among medicines the most valuable is cod-liver oil. It should be given even when the bowels are relaxed unless it causes an increase of diarrhœa, which is often not the case. Steel wine and quinine are also useful. I have often prescribed the *liquor ferri pernitratis* with advantage. Dr Eustace Smith has seen marked benefit result from the administration of tannic acid in doses of half a grain to a grain twice or thrice daily, as suggested by Dr Alison. If an occasional aperient is necessary, a little castor-oil, or a powder of rhubarb and soda, or the *liquor sennæ dulcis* may be given. I do not think that lime-water should be made use of as a matter of routine, nor unless there is proof that the milk which the child takes becomes curdled in its stomach.

Sir William Jenner has expressed strong objections to the administration of repeated doses of mercury to rickety children under any circumstances; and he says that when they are attacked with acute diseases leeches should never be applied, nor antimony be given to them.*

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* [Beside the discussion on rickets in the International Congress of 1881, referred to above, an instructive debate will be found in the 'Pathological Transactions' for the same year (vol. xxxii, pp. 312—404). It was introduced by the author of this work and continued by Dr Norman Moore, Mr Haward, Dr Dickinson, Mr Parker, Sir William Jenner, Mr Hutchinson, Mr Lucas, the late Dr Baxter, Mr S. Watson, and Dr Goodhart.—Ed.]

MOLLITIES OSSIUM

Nomenclature—Ætiology—Symptoms—Diagnosis—Histological and chemical changes—Pathology, &c.

In the middle of the last century instances in which extreme deformities had been produced by softening of the bones were recorded, almost in the same year by three observers, Duverney, Morand, and Pringle; and the names of two of the patients—the Marquise d'Armagnac and Madame Supist—have become historical. Similar cases have since been met with from time to time, but very rarely, except in certain districts bordering upon the Rhine, where, according to Senator (in Ziemssen's 'Handbuch') they have been somewhat less infrequent. The only one that I have seen was in a woman, aged forty-five, who died in Guy's Hospital under my uncle, Mr Hilton, in 1864. This case was related by Mr Durham in the 'Guy's Hospital Reports' for that year. The disease is called *Mollities Ossium*, *Malacosteon*, or *Osteomalacia*.

It is far more common in women than in men; among one hundred and forty-five cases collected by Mr Durham thirteen only occurred in males; and one may suspect that the disproportion would have been still more marked if they could have been sifted very critically. A definite cause can seldom be assigned to it. Habitual exposure to cold and wet, as from living in a damp house, has sometimes been supposed to give rise to it, but to many instances such an explanation could not be applied. The patients have often been well fed and in easy circumstances. Sometimes it has been noted that they had been affected with rickets in childhood, but in all probability this was a mere coincidence, for there is no other reason to suppose that the two affections are in any way related to one another. The occurrence of utero-gestation appears to play an important part in the ætiology of mollities ossium; in ninety-one of Mr Durham's cases it began during pregnancy, or shortly after childbirth.

The *age* at which it is least uncommon is between twenty-five and thirty-five; a few of the patients are said to have been under twenty years old, and a few over fifty.*

As a rule, the earliest symptom of the disease consists of pains in the trunk or in the limbs, which seem to vary in character in different cases, and which may appear to wander or fly about from part to part, so that they are usually supposed to be "rheumatic." The next thing may be that one of the bones breaks without cause, or during some slight effort, as in getting out of bed. Or a progressive change in the figure may be noticed, the body becoming short or stunted, the back rounded and distorted laterally, the neck stooping so that the chin may be brought close to the sternum. There is an extreme lassitude with disinclination for any kind of muscular exertion. The patient waddles in walking, and has to help herself with sticks or crutches. Presently she is obliged to take to her bed. She now becomes a most pitiable object. Her bones may show numerous fractures; and these remain unrepaired, the broken ends being merely surrounded by a soft callus, and forming so many false joints. Her limbs also become bent in the strangest way, perhaps one leg outwards and other inwards, according to the pressure to which they have been subjected while she is lying or half-sitting, propped up with pillows, and "all in a heap." Towards the last the softening of the bones may be so extreme that one can bend them backwards and forwards with but little force, and without injuring them; bringing, for instance, the foot round so as to touch the back or the head. The more superficial bones, even the cheek bones, can often be indented by the finger or they may feel like egg-shells, as if they had merely a thin layer of osseous material on their exterior. For a time the general functions of the body may appear to be but little interfered with; the appetite and the digestion may be good; and menstruation may go on naturally. Pyrexia may be occasionally present, but not to any marked extent. Ultimately, however, the disease ends fatally, either by exhaustion or more commonly from simple inability to breathe, the ribs being dragged inwards at each inspiratory effort, so that scarcely any air enters the lungs. Another frequent cause of death is the obstruction to parturition caused by distortion of the pelvis. Usually the brim acquires what is termed a *rostrate* character, the pelvic symphysis forming a sharp angle between two prominences due to the pushing upwards of the acetabula and the parts adjacent. Cæsarean section has often been necessary, and has often ended fatally for the mother. It is a point of great importance that mollities ossium seems often to advance step by step during successive pregnancies, the patient in the intervals regaining strength to a considerable extent, and even being able to get about to work. The duration of the diseaseis usually from four to six years; but sometimes it has lasted eight, ten, or thirteen years. Once, however, in a case recorded by C. Schmidt, it is said to have ended fatally in three months. In very exceptional instances it has been recovered from. Tonics and cod-liver oil are the medicines which appear to be most likely of service.

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* [In a well-marked case which I lately saw with Dr E. O. Day, the disease began about sixteen, and Dr Rehn has recorded one in an infant ('Internat. Med. Congr., 1881,' vol. iv, p. 59); so also has Dr Berry, of Manchester ('Brit. Med. Journ.,' 1884, vol. i, p. 213).—Ed.]

The *diagnosis* of mollities ossium is very easy at an advanced stage of the disease, but it must be borne in mind that mere brittleness of the bones is not sufficient to determine it. In old people, and in persons who have been long bedridden, the ribs and some other parts of the skeleton are apt to undergo atrophy, so that one can very readily snap them with the fingers. In the inmates of lunatic asylums this change seems to be particularly frequent. But even in young persons a somewhat similar state of *fragilitas ossium* is sometimes met with. Some years ago I saw a young man who was dying of bronchitis, and in whom a large number of the ribs were found to be broken, as the result of muscular efforts in coughing. He had at different periods of his life had fractures of many of his bones from very slight injuries; if I am not mistaken (for I have no notes of the case) one femur had given way when he was quite a child. Another affection which might perhaps be mistaken for mollities ossium is sarcoma, or even carcinoma, developing itself in a large number of the bones simultaneously, and causing their spontaneous fracture.

The fundamental distinction, however, between all these morbid states and that with which we are now concerned is that in it the bones are not only fragile but soft. After death they are found to be readily cut to pieces with a knife, and they may feel like india rubber or even like cheese. On section, the compact substance may have entirely disappeared, or it may be reduced to a very thin lamina beneath the periosteum, within which there may be nothing but a soft pulpy material. Or the cancellous tissue, instead of undergoing uniform absorption, may be hollowed out here and there into rounded or oval cavities. In the case which occurred at Guy's Hospital in 1864 this change in the bodies of the vertebræ was so striking that at first it almost seemed as though there were masses of some soft growth, perhaps myeloid in character, which had eaten away the bone. On the other hand, the calvaria was considerably increased in thickness, and had a homogeneous texture, which was compared by Mr Durham to softened pasteboard.

On chemical analysis the composition of the bones is found to be greatly altered. Different observers give different figures, but it may be roughly stated that the proportion of inorganic constituents is reduced to about 30 per cent. The carbonate of lime is said to be diminished in quality even more than the phosphate, and the character of the latter salt to be changed, the amount of lime being deficient in relation to the acids, so that it no longer forms the ordinary "basic" compound. C. Schmidt has also stated that gelatin is often absent. Still it is clear that the percentage given above represents not the definite actual constitution of osseous tissue affected with mollities ossium, but an average derived from some parts in which the change is extreme, and from others which perhaps deviate but little from the normal. As a rule, the morbid process seems to be most advanced in the interior of the bone, and least so towards the circumference. The microscope shows that there may be wide differences even within a single Haversian system, the lamellæ nearest the central vessel being completely decalcified, while the outer ones still retain their inorganic constituents. The orifices of the Haversian canals on the surface of the bone are widened, and a viscid fluid may exude from them when the periosteum is stripped off, which itself is very thickened and unduly vascular.

The substance which fills the interior of the bones in mollities ossium seems not to differ essentially from the normal medulla. It varies in appearance, being sometimes of a deep red colour, and spotted with ecchymoses, sometimes opaque, yellow, and fatty, sometimes mucoid and semi-translucent. These variations probably correspond with those which naturally occur in the same tissue. In the red material there are numerous cells, some of which may contain two or sometimes several nuclei. At least this is what Virchow and other histologists have generally stated. Rindfleisch maintains, on the contrary, that exceptionally few young elements can be seen.

With regard to the *pathology* of mollities ossium nothing as yet is certainly known. The decalcification of the bones has been supposed to be due to the action of some acid, and lactic acid is said to have been actually detected in the osseous tissue by several of those who have made analysis of it, and also in urine passed during life. In one case which ended in recovery Moers and Mück found that the acid gradually disappeared from the urine as the disease was subsiding ('Deutsches Archiv,' 1869).

Rindfleisch, on the other hand, suggests that the solvent is carbonic acid. Recent observers have failed to find in the urine an excess of phosphate and of carbonate of lime which was at one time said to be present, and, according to Mr Solly, in one case reached four times the normal quantity, such phosphatic deposits occurring in urine having an acid reaction (?). Phosphatic calculi seem, however, to have been found in the kidneys or in the bladder in certain cases.

It is a point of some interest that *arthritis deformans* has sometimes been noticed in connection with mollities ossium. For in the muscles also changes are found for which it seems not to be a sufficient explanation to refer them to mere disuse. Not only do they become flabby, wasted, and fatty, but Friedreich has recently shown that the nuclei of their fibres multiply and that there are other histological appearances identical with those which occur in progressive muscular atrophy. Trousseau and Lasègue are said to have observed in some cases that gently touching or stroking the surface of the limbs was capable of exciting painful contractions of the muscles beneath. It may therefore be that mollities ossium is, after all, something more than a mere disease of the bones.

DISEASES OF THE BLOOD

CHARACTERISED BY ANÆMIA AND HÆMORRHAGE

SCURVY

History—Early symptoms: purpura, spongy gums, hæmorrhagic inflammations —Night-blindness—Course and prognosis—Ætiology—Prophylactic treatment—Theory of the cause and symptoms—Curative treatment—Diagnosis.

History.—It is impossible to doubt, from what we know of the cause of scurvy, that men must have been liable to it ever since they began to live in masses under artificial conditions. But no positive proof of this can be found in the medical writings of antiquity, nor until the time of the Crusades. The name—which with slight modifications runs through the principal European languages—is said, by Immermann, to occur for the first time in the Botanologicon of Euricius Cordus (1534) in the German form "Scharbock," its root being a Danish word *Skörbeck*, signifying "disease of the mouth." Towards the end of the fifteenth, and in the sixteenth centuries, this complaint became very conspicuous from the ravages it committed among those who took part in the long sea-voyages which were then undertaken to the East round the Cape of Good Hope, and afterwards to America. But it was soon found that, beside the "sea-scurvy," there was a precisely similar "land-scurvy," which arose from time to time among the inhabitants of besieged towns, in the inmates of prisons and asylums, and among the poorer classes generally, especially when exposed to privation or famine. Unfortunately, however, the subject was afterwards thrown into complete confusion by the publication of a work 'De Scorbuto' by Severus Eugalenus (1588), in which the symptoms and effects of almost every disease were jumbled up together. He seems to have had many followers; and one of the results of their teachings still remains in the popular use of the word "scurvy" as a name for eczematous and other eruptions, and in the traditional practice, among medical men in country districts of England, of calling obstinate chronic sores upon the leg "scorbutic ulcers." Indeed, much of the voluminous medical literature of the seventeenth century shows so complete an ignorance of the real characters of the disease that Hirsch ingeniously argues that it cannot have been common. A reaction, however, at length occurred, with Sydenham for one of its leaders; and in 1753, Dr James Lind, Physician to His Majesty's Royal Hospital at Haslar, gave an admirable description of *Scorbutus* or Scurvy, which has been followed by all later writers.

Onset and early symptoms.—Usually, but not always, the more definite symptoms of the disease are preceded by a general failure of health and strength. The face becomes pale and sallow, with a livid discolouration of the lips and cheeks. So characteristic is the patient's appearance that its cause may often be known at a glance. When several are attacked at the same time, each is struck with the altered aspect of the others, while unaware that he presents the same change. The skin is dry and scurfy, and its hair-follicles are prominent and rough to the touch, as with "goose-skin" or with *pityriasis tabescentium.* The spirits are depressed and gloomy, and the mind apathetic and indifferent, with great lassitude and a sense of fatigue and shortness of breath after exertion. The muscles waste and are soft and flabby. There is much sensitiveness to cold; and pains, which are commonly spoken of as rheumatic, are experienced in different parts of the body, but especially in the loins and in the calves of the legs. These pains are worse after exertion, and are relieved by rest and sleep.

A week or two later a purpuric eruption is seen upon the skin, generally first over the lower limbs, but afterwards on the arms and on the trunk; not often upon the head or face. It consists of reddish or purple spots, chiefly of small size, and presenting the peculiarity that most of them have a hair or a hair-follicle in the centre. The projecting state of the follicles may cause these spots to be slightly raised above the surface. In severe cases there may also be vesicles or even bullæ, containing a sanguineous fluid, and they may presently dry up into crusts, or their bases may ulcerate. Much more common are large subcutaneous extravasations of blood, constituting what are termed *vibices,* the edges of which are ill-defined, fading off with varied tints: these sometimes break down, and form larger ulcers, with spongy floors, exhaling a thin, bloody, putrid fluid. One or more of the nails may be detached by effused blood from its bed, and may be cast off by an ulcerative process at its root.

Another very characteristic symptom is the formation of ill-defined brawny indurations in the connective tissue, especially of the hams, but also behind the ankles, along the backs of the thighs, over the recti abdominis, in the armpits and elsewhere. The skin over them may be free from discolouration, but they nevertheless consist of extravasated clotted blood, mixed perhaps with gelatinous inflammatory exudation. They are sometimes very rapidly developed, and may be hot and painful.

Again, blood is often poured out into the substance of the muscles, forming more or less obvious swellings, and rendering their contractions painful and difficult. Or it may detach periosteum from the bones over a more or less extensive area. This is most frequent along the front of the tibia, where an enlargement results which has sometimes been mistaken for a syphilitic node. A similar change may also affect a rib, or the scapula, or one of the jawbones, and may sometimes lead to superficial necrosis and to exfoliation. In yet other instances, extravasation takes place along the epiphysial lines of growing bones, and causes their separation. Or, if there should have recently been a fracture which has undergone repair, the callus may soften down, and the broken ends may again become loose. Or effusion of blood may take place into some of the joints, especially the knees and ankles.

But the most remarkable affection of all, and one which is scarcely ever absent, is that of the gums. Their edges become bluish-red, spongy, and detached from the teeth, with which they should be closely in contact. They are also painful and tender, and they bleed at the slightest touch. They may even become black, and so greatly swollen as to rise above the level of the teeth or even to protrude from between the lips. This change is most marked opposite the incisors, but it may also extend round towards the molars. When there is a gap among the teeth, the corresponding part of the gum remains healthy. And in young infants, as well as in toothless old people, the gingival affection is said to be altogether wanting. M. Fauvel, at the Salpêtrière, had in 1847 a case in an old woman in whom a single remaining tooth was surrounded by a mass of swollen gum; the tooth was extracted, and the gum soon became level and firmer. When it is severe, a scorbutic state of the gums often renders mastication and the ingestion of solid food an impossibility. There is a horrible fœtor of the breath. The teeth become loose and may fall out. Very commonly a greyish diphtheritic layer forms upon the surface of the affected parts, and the gums may slough, so as to expose the alveolar processes. It is a curious circumstance that the mucous membrane of the rest of the mouth remains perfectly healthy, or at most is slightly livid and puffy. MM. Lasègue and Legroux, however, speak of having frequently seen ecchymoses on the palate during the epidemic which accompanied the siege of Paris in 1871. The tongue is large and marked by the teeth at its edges.

Hæmorrhages from mucous membranes are of frequent occurrence in scurvy, epistaxis being especially common. Bleeding may also take place from the stomach and intestines. In many epidemics dysenteric symptoms are very constantly present, but this appears to be due to a coincidence of the two diseases. Blood seems not to be often expectorated from the lungs, except when gangrenous pneumonia sets in as a complication, as is sometimes the case. More frequently the pericardium, or one of the pleural cavities, or both together, are attacked with an inflammatory process, attended with an abundant escape of blood as well as with the usual inflammatory exudation.

I have said that the face does not often present purpuric spots in scurvy. But the skin round one or both of the eyes sometimes becomes puffed out into purple swellings, while the conjunctiva covering the eyeball assumes a brilliant red colour and projects far above the level of the cornea. Dr Buzzard, in 'Reynolds' System,' says that in many cases seen by him during the Crimean War, this condition (which was not inflammatory, being attended with neither pain nor discharge) constituted the chief symptom of the disease, and that they were usually very severe cases and often ended fatally. But sometimes hæmorrhage is said to occur into the interior of the eye, and especially into the anterior chamber; this condition may be complicated with iritis. Or there may be hæmorrhagic choroiditis, or even panophthalmitis with sloughing of the cornea.

But the most curious symptom of the disease is what is known as "hemeralopia," "nyctalopia," or "night-blindness." The patient can see well during the day, and at night he can distinguish objects near a candle and even read. But when he has not the assistance of artificial light, he becomes so blind, even though the moon may be shining, that he has to be led about. The pupils may be dilated in such cases; but there are no ophthalmoscopic changes. In vol. ii of the 'Ophthalmic Hospital Reports' (1859), papers on this subject will be found by Dr Bryson and others, founded upon observations made during the Crimean War, and in Her Majesty's ships in different parts of the world. Not infrequently a sudden night-blindness has been the earliest indication that the patient was otherwise than well.

It is to be observed that the order in which the various symptoms of scurvy develop themselves is very different in different cases. Sometimes the gingival affection is the first to appear; it may even constitute the sole manifestation of the disease; but sometimes it follows the purpura by a considerable interval. There may have been no sign of the patient's being otherwise than well until some part of the skin, which has received a trifling blow, becomes the seat of extensive extravasation of blood, or an ordinary purgative dose may, quite unexpectedly, be followed by profuse intestinal hæmorrhage, or an old chronic ulcer of the leg may be found to be spongy and the discharge from it sanious.

Course and event.—The course of scurvy is slow and protracted. There is no fever, except, perhaps, when there are inflammatory complications. The appetite is bad, but sometimes there is said to be a longing for vegetables and fruit. Thirst is often marked. The pains in the limbs become so severe as to interfere with sleep. Anæmia and emaciation advance rapidly, and subcutaneous œdema is present in many cases. The urine is sometimes albuminous, even though the kidneys may be subsequently found to be healthy. The pulse becomes extremely small and weak, and the heart's impulse may be imperceptible. The muscular weakness is frequently so great that the patient faints if he attempts even to sit up in bed. Indeed this is sometimes the cause of a fatal termination at a comparatively early stage of the disease, which otherwise seldom destroys life, even in bad cases, until after the lapse of some weeks. Thus it has happened that men have suddenly died while being moved into hospital, and at the old Dreadnought it used to be a rule that all those who were ill with scurvy should be hoisted up the ship's side in a recumbent position. It is rare for hæmorrhages from mucous membranes to destroy life directly. Death is more often due to gradual exhaustion and prostration, and then the mind is usually clear to the last. In other cases it is the result of some complication, such as dysentery or croupous pneumonia (which may or may not pass into gangrene) or ulcerative endocarditis; or, again, it follows extravasation of blood into the cerebral membranes, or the hæmorrhagic forms of serous inflammation to which I have already adverted.

But in the immense majority of cases—at least when the patient comes under medical observation—recovery takes place. The improvement produced by proper treatment is often immediate and very striking; but nevertheless many weeks or even months pass before the health is completely restored.* The purpuric spots undergo changes of colour like those which are seen in bruises, and gradually disappear; the smaller ones round the hair-follicles merely turn brown. The brawny indurations slowly subside, but they not unfrequently leave behind them thickenings and fibrous bands which may cause permanent contractions of parts of the limbs, especially at the knee or the ankle, with atrophy of the corresponding muscles. Even the joints themselves may be reduced to a state of ancylosis.

Cause.—Our knowledge of the ætiology of scurvy is so far perfect that we habitually prevent its occurrence under circumstances that would inevitably give rise to it but for our interference; and there are very few diseases of which as much can be said. Still, several points require to be carefully and separately discussed. One is whether the chief or ordinary cause of scurvy, which we have thus under our control, is the cause of all cases without exception. Now, beyond all doubt that cause is the absence in the dietary of a due supply of *fresh vegetable food.* The question therefore is whether scurvy ever arises when a proper quantity of such food has been taken. I am very much disposed to think that to this a negative answer may be given. Immermann, indeed, cites in Ziemssen's 'Handbuch' a few instances in which he says that the disease prevailed notwithstanding that there was an abundant supply of vegetables. But on looking up the first of them, an epidemic which occurred in the barracks of Rastatt in the winter of 1851—52, I find that Opitz, who recorded it nine years later in the 'Prager Vierteljahrschrift,' says expressly that the rich lived almost entirely on soup and beef and dumplings because vegetables were very scarce and dear. And even had they been cheap, it would have proved nothing, unless those persons who were actually taken ill could be shown to have partaken freely of them. So, again, it is not sufficient to assert that vegetable food has been duly served out in rations to a body of men, some of whom are found to have scurvy; we want to know that they have eaten it, and have not thrown it away, or parted with it to their comrades. And I am not aware that any case is on record in regard to which these conditions are satisfied. It is to be noted that potatoes have a high anti-scorbutic value; the disease was exceedingly prevalent in Ireland after the failure of the potato crop in 1846, and at Millbank Penitentiary in 1823 an outbreak occurred which was directly traced to the introduction, a few months previously, of a new dietary from which the potato was omitted. On the other hand, peas and beans are incapable of preventing scurvy, and the same is the case with rice and other cereals. There is no reason to doubt that the modern methods of presevering vegetables in a succulent condition leaves them with their antiscorbutic properties almost intact; but complete desiccation seems to destroy their usefulness. All fruits, including apples, tend powerfully to ward off the disease; but cider is said to have no such power. The acid wines of France, however, are believed to be antiscorbutic.

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* [Dr Carrington tells me that at present these protracted cases are not seen at the Seaman's Hospital ("Dreadnought") at Greenwich, and that during six years no permanent injury has followed scurvy. In the worst cases the rations have been short and the water bad.—Ed.]

But it is undoubtedly the fact that scurvy does not always show itself in those who fail to receive a proper share of vegetable food. And so we have to ask, in the second place, whether the disease requires accessory causes for its production; or, if not, what circumstances are capable of counteracting its chief cause. Now, there are some conditions which affect the frequency of the occurrence of scurvy, and which are mentioned by writers on its ætiology, but which really concern it only indirectly. Thus it is more common in cold climates generally than in hot ones, in winter than in summer, only because a low temperature is unfavourable to the growth of plants. Still, there are grounds for believing that the development of the complaint is favoured by a variety of depressing influences, among which may be mentioned want of sunlight, residence in narrow, cold, dark, damp dwellings (such as cellars or the casemates of a fort), over-fatigue (especially after a long period of comparative inaction), excessive indulgence in ardent spirits, despondency of mind, &c. It is also said to be particularly apt to occur in persons who are convalescent from ague or dysentery, in those who have syphilis, and in soldiers who are recovering from severe wounds.

On the other hand, it is certain that the withdrawal of fresh vegetables from the dietary can be neutralized in several different ways. Thus fresh meat, if eaten in large quantities, is an "antiscorbutic," especially when raw or but slightly cooked. Indeed, one of the earlier views with regard to the ætiology of scurvy was that it was a direct effect of the salt pork which constitutes so large a part of the diet of sailors; but it has often occurred when no salted provisions of any kind had been taken. With regard to the antiscorbutic value of milk, statements have been made which are not altogether in accord with one another. The matter is discussed in a valuable essay by the late Dr Parkes in the 'Med.-Chir. Review' for 1848. The conclusion seems to be that the ingestion of milk in quantities of a pint or a pint and a half every day does not always supply the omission of vegetables in preventing scurvy; but children and others who live mainly on milk undoubtedly remain free from the disease. On the other hand, infants suckled by scorbutic mothers have often been attacked; and it is very likely that the milk of cows fed almost entirely upon hay may fail to possess the same properties as that of animals which have had plenty of grass.

The most important of all antiscorbutic agents, however, are, in the absence of fresh vegetables, the juices of certain fruits, especially the orange and lemon. Their value was recognised as far back as 1573, by Solomon Albertus; and since 1795 *lime-juice* has been regularly furnished to ships in the Royal Navy, with the result that scurvy, which used to commit the most fearful ravages among the sailors, is now scarcely ever seen. Indeed, were it not for the systematic use of this agent, every long voyage, when vegetables are no longer to be had, would probably be an experiment demonstrating the real cause of the disease. The usual plan is to serve out an ounce of the juice daily to each man. In the spring of 1876 an outbreak of scurvy took place among the men of the sledging parties sent out from the ships "Discovery" and "Alert" engaged in the Arctic Expedition. These men had no supply of lime-juice with them, and received only very small quantities of potatoes.* Nevertheless it was afterwards argued that the defects in their dietary were not the cause of their falling ill, chiefly on the ground that cases arose within from ten to twenty-seven days after the commencement of the sledging operations, and must consequently have had their origin in the unfavourable conditions under which the men had laboured during the previous long winter. But the Admiralty Committee appointed to inquire into the matter reported that in their opinion the disease was due to the absence of lime-juice. In future, it would, under similar circumstances, be well to concentrate the juice so as to render it more portable; and glycerine might be added to prevent freezing.

Why the absence of vegetables from the food should cause scurvy, and how lime-juice is able to take their place and to prevent the development of the disease, are questions upon which there has been much speculation, but hitherto with no positive result. In the last century dilute sulphuric acid and vinegar were largely made use of, in the vain hope that they too might prove to be antiscorbutics; and crystallized tartaric and citric acids have since been tried and have generally failed.

Dr Garrod, in 1848, propounded the theory that the essential cause of the disease was the absence of a due supply of potass in the food. He showed that there was a great deficiency of the salts of this alkali in dietaries which were known to be liable to give rise to scurvy, and that it was present in abundance in all the substances which possessed antiscorbutic properties. It is curious that whereas his views have never been widely accepted in this country, they are with a slight modification upheld in Germany by the most recent writers at the present time. The modification, which was (I believe) first suggested by Chalvet, consists in rejecting as valueless those potass-salts that pass out of the body unchanged, and in attaching importance solely to such of them as undergo conversion into carbonates and may fairly be supposed to be capable of entering into the composition of red blood-discs or of muscle. It has in fact been demonstrated that nitrate of potass is incapable of preventing scurvy. But, on the other hand, I am not aware that anyone has yet shown that the citrate or the tartrate of potass, in a state of purity, possess antiscorbutic value at all comparable with that of lime-juice or fresh vegetables.

Histology.—There can be little doubt that the immediate cause of the purpura and of the other hæmorrhagic symptoms of scurvy is a morbid condition of the walls of the smaller vessels. But hitherto no visible change in them has been detected, even with the microscope. In 1871 Lasègue and Legroux examined the capillaries in seven fatal cases which had occurred during the epidemic in Paris at the time of the siege; they could discover nothing but some scattered fat-granules. Nor have we as yet any certain knowledge as to the alteration in the composition of the blood which must be supposed to form an intervening link between the dietetic cause of the disease and its varied phenomena. After death the blood within the body has sometimes been found to be coagulated; sometimes it has been liquid. It has not seldom been pale and watery; but this is only equivalent to saying that anæmia is a symptom of scurvy. For the same reason it is doubtful whether much importance can be attached to observations proving that the red discs are deficient in number. Such oligocythæmia necessarily involves a deficiency of potass salts and of iron, which has in fact been shown to occur by several chemists. Laboulbène (1861) made out a slight degree of leucocytosis; but this again may probably have been merely relative, depending upon the scarcity of red discs.

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* [They had been given extra rations of lime-juice for some time before starting in order to saturate their systems! No case of scurvy from the Royal Navy has been admitted into the Seaman's Hospital since 1879. Most of the cases there treated occur in Norwegian sailors.—Ed.]

Dr Garrod, Dr Ralfe, and others have carefully investigated the state of the urine, in the hope of indirectly throwing light upon the constitution of the blood in scurvy. Dr Ralfe's conclusions are that the uric acid is increased, but that the acidity of the urine is diminished, and that there is a great reduction in the amount of alkaline phosphates. And he has propounded the theory that the primary change in the blood is a diminution in its alkalinity, citing in support of this view certain experiments upon animals by Hoffmann and others, in which it has been shown that food yielding only an acid ash produces, after a time, effects comparable with the symptoms of scurvy, namely dissolution of the corpuscles, ecchymoses, and purpura.

Treatment.—In the treatment of persons already ill with scurvy, the administration of lime-juice still plays the most important part. The presence of diarrhœa must not be supposed to contraindicate its use.

If solid fresh meat, mashed potatoes, cabbage, and salad can be eaten, there is no objection to the patient's having them; but when the gums and teeth are very tender, the diet often has to be limited to milk, beef tea, and eggs beaten up with wine. On the Continent the yeast of beer is very highly spoken of.

Dr Buzzard says that a daily application of solid nitrate of silver to the gums affords great relief when they are sloughing and bleeding. Washes of chlorine, Condy's fluid, alum, decoction of oak-bark, may also be freely employed. For the hard swellings in the legs friction with soapsuds and water is said to have been used with success in the Turkish hospitals in the Crimean War. Iodide of potassium is recommended when effusion occurs under the periosteum of the bones. It is advised that scorbutic ulcers should be dressed with lint soaked in lemon-juice, or with the bruised substance of succulent herbs, such as the house-leek.

Diagnosis.—I have been obliged to leave to the last the consideration of the diagnosis of scurvy, because the ætiology of the disease plays so important a part in it. Most observers, indeed, are of opinion that, assuming it to be a positive fact that an individual has eaten an adequate amount of fresh vegetable food, one is justified, on that ground alone, in denying the scorbutic origin of any complaint from which he may be suffering, however closely his symptoms may resemble those of scurvy.

A case in point was brought by Dr Stephen Mackenzie before the Ophthalmological Society in 1880. It was that of a lad who was extremely anæmic, and who had cutaneous purpura and spongy gums, but who (it was stated) had not been deprived of vegetable food. There were, however, hæmorrhages in each retina, with patches of degeneration like those which occur in Bright's disease. This in itself is a reason for thinking that the complaint was probably not scurvy, for in that complaint retinal effusions of blood are very rare.

As a rule, the gingival affection constitutes a safe criterion between scurvy and other purpuric affections, including the "morbus maculosus."

But in splenic leuchæmia Mosler and others have sometimes found the gums swollen and ulcerated, as well as inclined to bleed. And in some cases of malformation of the heart I believe that slight morbid conditions of the gums have been seen which might have been regarded as scorbutic. But whenever there is a doubt as to the diagnosis, it may be quickly settled by the administration of lime-juice. And the same may be said with regard to the exceptional cases of scurvy in which the gums remain in a normal state, as well as of those other rare instances in which there are no symptoms except the gingival change.

But the most important point of all is the danger of overlooking the slight forms of scurvy occurring sporadically, as, for example, among the poor of London and other cities. Dr Buzzard has especially insisted on the fact, that such persons, who probably never brush the teeth, attach no importance whatever to an unhealthy condition of the gums. Nor do they notice a slight petechial eruption upon the legs. What they are likely to seek relief for is a supposed "rheumatic" complaint attended with muscular weakness. It is essential that one should be on the look-out for the other symptoms of scurvy, including the peculiar sallow complexion, when one's advice is thus asked for vague pains in the limbs. In my out-patients' practice at Guy's Hospital I remember more than one case in which I was able to detect the disease in men who had in fact been living without vegetable food, although they had not the slightest idea that their symptoms were attributable to that cause until I told them so.

ANÆMIA

General description of anæmia—Amount of blood—Number of red corpuscles—Amount of hæmoglobin—Changes in the blood-discs—Anæmic murmurs—Dyspnœa—Fatty degeneration of the heart—Other symptoms—Symptomatic and essential anæmia.

CHLOROSIS—*Its relation to menstruation—to imperfect development of the heart and vessels—Symptoms—Prognosis.*

PERNICIOUS ANÆMIA—*History—Name—Age and sex—Antecedents—Course and symptoms—Pathology—Prognosis—Treatment.*

PARASITIC ANÆMIA—*The ankylostomum duodenale and Egyptian or tropical anæmia—Natural history of the worm—Symptoms, prognosis and treatment.*

Under various circumstances the tissues of the human body which normally appear to have a pink or red hue, from the blood circulating through them, become pale or even white, as a result of deficiency of blood or of the colouring matter it contains. There is, indeed, great difficulty in fixing the boundary line between health and disease in this respect. Some persons—nearly all the members of certain families—are naturally pale, and remain so always, however favourable may be the conditions under which they live. Others lose their colour from time to time if they reside in large towns, work at sedentary occupations, or keep late hours; but a rosy complexion returns soon after they go back to the country, under the influence of sunlight, fresh air, and exercise. We may therefore conclude that among those who constantly exhibit pallor of countenance, but who are all their lives confined to dark workshops, or cellars, or mines, some, at least, would assume a very different appearance if they could spend their time in light well-ventilated rooms or in the open air. But, again, there are others who owe their colourless, sallow appearance to habitual excesses, especially to the practice of onanism, or to sexual indulgence at too early an age. And, for my own part, though I doubt whether moderate smoking is injurious to health, I should be disposed to reckon the abuse of tobacco as among the most important causes of a similar condition; though whether it acts directly or by disturbing the digestive organs I do not know. Again, the skin becomes pale as the result of diseases of almost any kind, whether acute or chronic. Another frequent cause is the occurrence of hæmorrhage to a very considerable extent or repeated at short intervals. Lastly, there are cases for which no explanation whatever can be offered except that the blood-forming organs are for some reason or other unequal to the calls made upon them, and that the circulating fluid is consequently impoverished.

The name which is now universally applied to the state in question is Anæmia, and for all practical purposes it is sufficiently accurate, so that there is no reason to regret the failure of an attempt which was made some years ago to substitute for it that of "oligæmia." It is characterised by pallor not only of the skin but of all visible mucous membranes. However, as I shall afterwards point out, the tint varies widely in different cases, and sometimes, instead of being white like marble, it is very decidedly yellowish. The hands, the fingers, the finger-nails, show the change as distinctly as the countenance: or even more so, for in those persons who have the smaller vessels of the cheeks dilated and varicose there may remain a crimson patch on each side of the face, contrasting strangely with the rest of the complexion. The lips, the tongue, the fauces, the lachrymal carunculæ, are all more or less white and waxy looking. So, again, in the dead body, the amount of blood in the deeper tissues and in the various organs is found to be very deficient; the liver and the kidneys look like wax; the heart and the great vessels may contain only small loose shreds of coagulum and a little thin pink fluid.

Estimate of amount of blood.—The evidence derived from post-mortem examinations seems to me to show beyond question that the total volume of the blood is greatly diminished, at least in the more severe cases of anæmia. But, unfortunately, it is difficult to obtain clinical proof of this fact, and almost impossible accurately to estimate the degree of deficiency. The only method seems to be that proposed by Quincke, of first counting (by a process presently to be described) the proportion of red discs contained in the patient's blood, then transfusing into his circulation a known quantity of healthy blood, and soon afterwards again counting the red discs in a fresh specimen taken when the new blood and the old may be supposed to have become thoroughly mixed together. A simple formula gives the total volume of blood which was present in the patient's body before the operation. In two cases of "pernicious anæmia" Quincke estimated by this method that the blood formed only 4 or 5 per cent. of the body weight instead of 7·7 per cent., which is supposed to be about the normal proportion.

Amount of corpuscles.—Such experiments, however, can rarely be carried out accurately enough to yield results that can be relied on. And for practical purposes we may be content to remember that diminution to whatever extent of the total volume of the blood directly corresponds with certain changes in its composition which can be estimated with comparative facility.

One of these changes is, in most if not in all cases, a diminution in the proportion of red discs to that of the liquid in which they float. The "numeration of blood-corpuscles" is one of the most important advances in practical medicine that has been made within the last few years. Originally suggested by Vierordt, it has been simplified by subsequent investigations, and especially by Malassez (1872) and by Gowers (1877), until it has now become a very easy matter. The principle is to dilute to a definite extent a measured quantity of blood, and then to count the number of red discs contained in a certain volume of the mixed liquid. The method of employing the *Hæmocytometer* of Gowers is as follows: (1) A small pipette, holding exactly 995 cubic millimetres, is filled with the diluting solution, which is then poured into a small glass jar or mixing vessel. A good solution, which leaves the corpuscles in a state favourable for observation, consists of sulphate of soda in distilled water of sp. gr. 1025. (2) The The patient's finger is pricked with a lancet, so that a drop of blood escapes without much pressure. Five cubic millimetres of blood are then taken up by a fine capillary tube, graduated for the purpose, and are blown into the diluting fluid in the vessel. If there is a difficulty in getting exactly the right quantity of blood into the tube the best way is to take up a little more than enough, and then to let the excess escape into a soft cloth. (3) The contents of the mixing-jar are well stirred up with a glass rod. (4) A drop of the mixed liquid is placed in the centre of a cell excavated in a microscopic slide. This cell is exactly one fifth of a millimetre deep, and its floor is ruled in tenth of millimetre squares. The slide rests on a metal slip, to which two springs are attached. (5) A cover-glass is next laid over the cell, in contact with the liquid in it; the springs are brought over the edges of the cover-glass, and keep it in position with a pressure which is always uniform; the slide is placed horizontally on the stage of a microscope, and this is focussed upon the squares in the floor of the cell. (6) In a few minutes the red discs are found to have settled down upon the squares by gravitation. The number in the squares is now counted, and this, multiplied by 10,000, gives the number contained in one cubic millimetre of the blood. The average number in normal blood is believed to be 5,000,000 in the males and 4,500,000 in females. It is usual, however, to state the "corpuscular richness" of blood as a decimal fraction of the normal richness, this being taken at 5,000,000 to the cubic millimetre. The decimal figure may be obtained by dividing by 5 the number of leucocytes contained in 10 squares. Thus, in a case of Dr Gowers, the number in 10 squares was 332; the "corpuscular richness" was therefore ·66.

Amount of hæmoglobin.—It is now known that the deficiency of red discs in proportion to *liquor sanguinis* after all affords an incomplete measure of that which is believed to be the fundamental change in the composition of the blood in anæmia, namely, the diminution in the amount of hæmoglobin contained in it. For all practical purposes this may be estimated with sufficient accuracy by an apparatus called a *Hæmochromometer*, for which, in its most improved form, we are again indebted to Dr Gowers: he described it in the 'Transactions' of the Clinical Society for 1879. It consists of two glass cylinders of equal diameter, which are placed side by side upon a small wooden stand. One of them is closed, having been filled with glycerine jelly, coloured by a mixture of carmine and picrocarminate of ammonia, so that its tint is that of blood diluted with water in the proportion of one part in a hundred. The other cylinder is graduated in such a manner that a space equal to two cubic centimetres has 100 divisions. It is open, and empty except that a little distilled water is poured into it. Some of the blood the hæmoglobin of which is to be estimated, is now taken up by a capillary pipette, marked for twenty cubic millimetres; this quantity is carefully measured off, and is ejected from the pipette into the open cylinder, which is quickly shaken so as to secure the admixture of the blood with the water previously contained in it, before coagulation has had time to occur. Distilled water is then added, drop by drop, by means of a pipette-stopper, until the tint of the diluted blood becomes the same as that of the standard in the closed cylinder. The degree of dilution, when this point is reached, indicates the percentage proportion of hæmoglobin in the blood under examination, as compared with that of normal blood. The best way of observing the tint is to hold the apparatus up between the eye and a window, so that the light passes directly through the cylinder. The value of hæmoglobin for each red disc may of course be obtained by combining the results yielded by the hæmacytometer and by the hæmochromometer. Thus the blood of an anæmic patient of Dr Gowers contained 60 per cent. of corpuscles, but only 30 per cent. of hæmoglobin; the average value of red discs was of course $\frac{30}{60}$ths, or one half of their normal value. Not unfrequently it falls as low as one third.

It is to be observed that the deficiency of colouring matter in the blood of an anæmic patient is often obvious to the naked eye when the finger is pricked so as to allow a drop to be taken for investigation. One sees at a glance that it is pale, thin, and watery looking, exactly as if it had been diluted.

Corpuscular changes.—More or less marked alterations in the microscopical appearance of the red discs of the blood may also be made out in many cases. Thus, the average diameter of a normal corpuscle being 7·5 μ (1 $\mu = 0·001$ mm.), the average diameter in anæmia has been found by Hayem and by Eichhorst to be reduced to 7 μ, 6·5 μ, or even 6 μ. Moreover, red discs are sometimes present which are far smaller than any that exist in health, their diameter being from 6 μ to 2 μ; these have been termed *microcytes.* Contrasting with them, however, there may be others which are larger than normal, having a diameter of 12 μ; they have been called *megalocytes.* Lastly, Quincke and others have found red discs presenting curious irregular forms, being oval, elongated, curved, or drawn out into pointed processes. And another change that seems to have been first recognised by Drs Mackern and Davy, then students at Guy's Hospital, is that the hæmoglobin is sometimes separated from the substance of the corpuscle, forming a rounded body, which had been mistaken by previous observers for a nucleus. For these conditions the term *pœkilocytosis* has been very unnecessarily coined. They were at first supposed to be peculiar to a special affection which will be presently described under the name of pernicious anæmia; but they have since been discovered in cases of anæmia secondary to phthisis, to cancer of the stomach, or to chronic disease of the kidney.

Thus we probably may conclude that the most essential character of the blood in anæmia is a deficiency in the amount of hæmoglobin contained in it. We can even imagine that when the "corpuscular richness" of the blood is diminished, this may sometimes be due, rather to the want of an adequate supply of hæmoglobin for them, than to an actual failure of the organic process by which they are developed; but that the latter occurs in certain cases can nevertheless hardly be doubted. Again, it is obvious that there are two ways in which the amount of hæmoglobin may be reduced below the normal standard; (1) it may escape from the vessels by hæmorrhage, or be consumed within the body more rapidly than it can be reproduced; (2) the formation of it may be defective.

Now, it seems pretty clear that in those cases of anæmia resulting from hæmorrhage in which the patient quickly regains his colour as soon as the bleeding is arrested, all the formative processes, both chemical and histological, are in a perfectly normal condition. On the other hand, most pathologists have supposed that when anæmia seems to arise spontaneously as in the "pernicious" form of the disease, it is entirely due to a defect in these processes. But Quincke has lately recorded some observations which suggest the possibility that even in such cases there may be an undue destruction of red discs and of the hæmoglobin which they contain. He has found that in some instances the amount of iron in the liver is from ten to thirty times as great as under normal circumstances, and that there is also an increase of it, but to a less extent, in the kidneys and in the pancreas. The liver-cells present granules, which are supposed by him to consist of an albuminate of iron; they give to the organ a yellow brown colour which he seems to think characteristic; the addition of sulphide of ammonium turns the tissue of a greenish black colour; that of ferrocyanide of potassium turns it blue. However, the significance of this discovery still remains doubtful. Quincke himself has suggested that it may possibly have resulted from the medicinal administration of iron at some former period of the patient's life. A similar deposit of iron has also been found in diabetes and in enteric fever.

Murmurs.—Among the effects of anæmia, one which attracted great attention from the earlier auscultators was the production of abnormal bruits in the heart and in the great vessels. Thus when the stethoscope is lightly laid upon the patient's neck, just above the inner end of one clavicle, there is often heard a murmur of extraordinary loudness, which is continuous, but may present variations of intensity, corresponding with the cardiac or with the respiratory movements. This, from its humming quality, was by Bouillaud termed "bruit de diable," the "diable" being a toy common in Paris in 1835, which made a similar noise. There is no difficulty in proving that its seat is in the cervical veins, for one can instantly arrest it by laying one's finger transversely across the sterno-mastoid muscle above the stethoscope, and exerting gentle pressure. And, according to the theory which refers all murmurs to the formation of a "fluid vein," the *bruit de diable* may easily be accounted for, at least if we admit that in anæmia the volume of the blood is diminished. The sinus of the external jugular vein is fixed by adhesions to the cervical fascia. Consequently, when in an anæmic patient the veins in general shrink and adjust themselves to the small quantity of fluid circulating through them, this sinus remains unaltered in size, and forms a relatively wide space, within which the streams that enter it are thrown into vibration. The explanation is corroborated by the fact that in many healthy persons one can temporarily generate a *bruit de diable* by pressure with a stethoscope in the region where it occurs. All that is necessary is to narrow one or more of the veins that open into the sinus, and so to disturb the normal relation between them and it.

An "anæmic murmur" of another kind is systolic in rhythm, and is heard over the heart and the main arteries. It is usually loudest at the base, and it often seems to be traceable along the pulmonary artery rather than along the aorta. This murmur can be accounted for in the same way as the other. The bases of the two main arteries are supposed to be unable to retract, in correspondence with the diminished volume of the blood, to the same extent as the orifices through which the blood enters them. Whether an anæmic murmur is ever localised at the apex I am not sure. Writers on auscultation say that this may be the case; but the question is difficult to answer, on account of the doubt which prevails as to the origin and nature of apical bruits occurring even in persons who are florid.

It is important to notice that none of these murmurs are of the slightest significance, from a clinical point of view, so far as the diagnosis of anæmia itself is concerned. But they are of considerable importance because they would almost certainly be regarded as signs of organic disease of the heart or of the great vessels by anyone uninformed as to their characters who should discover them for the first time. This is especially the case with the basic systolic murmur, which often has a rough harsh quality suggestive of anything rather than a functional origin. And when there are other reasons for suspecting an organic affection of the heart, as, for instance, when the patient has had rheumatic fever, it is often very difficult to determine whether such an affection may not be present, and whether the anæmia may not after all be merely one of its effects instead of being the primary disease.

Dyspnœa.—Considering that the red discs of the blood have the function of carrying oxygen to the tissues one is not surprised to learn that a disturbance of the respiratory processes is among the most marked effects of anæmia. Dyspnœa is almost always present; even when the patient is at rest the breathing is unduly rapid without his being conscious of it; when he makes any effort he may be seized with the most distressing suffocative paroxysms and with palpitation of the heart. In some cases similar paroxysms come on without apparent cause. According to Immermann it is difficult to give a completely satisfactory explanation of these facts, but I should have thought it sufficient to suppose that the nerve-cells of the respiratory centre, which are known to be stimulated to excessive discharge by blood containing a normal quantity of hæmoglobin when this is imperfectly oxygenated, are affected in precisely the same manner by blood in which the hæmoglobin is greatly diminished in amount, notwithstanding that what there is of it may be saturated with oxygen. In either case the oxygen that reaches the nerve-cells is deficient.

Fatty degeneration.—However this may be, there can be little doubt that the reduction in the amount of oxygen supplied to the various tissues is the cause of one of the most striking of the morbid appearances which are found in the bodies of those who have died in a state of extreme anæmia, namely, a granular or fatty degeneration of the muscular substance of the heart as well as of the lining membrane of the larger vessels, and of the secreting cells of the gastric glands, the liver, and the kidneys. At one time we supposed at Guy's Hospital that such changes were peculiar to the form of anæmia which had been described by Addison, and which is now known as pernicious anæmia. But in 1873 the characteristic appearance of the heart was observed in a woman who died of cancer of the breast; in 1874 in a woman who had had hæmatemesis from an ulcer of the stomach; and in 1877 in a man who had suffered severely from hæmaturia, and, again, in a woman who had had a bleeding malignant tumour in the neck. The degeneration is not universally distributed, but specially affects the muscular fibres of the columns of the mitral valve and those which lie beneath the endocardium lining the septum and the ventricles generally. It gives rise to the formation of a series of parallel opaque-yellowish or cream-coloured lines, which run across the direction of the fibres themselves, and to which we have been accustomed to allude under the name of the "tabby-cat-striation" (p. 38). With a microscope the opacity and pallor are seen to be due to the presence of closely aggregated fat-granules and globules, which look black by transmitted light. They are doubtless waste products that have arisen during the generation of the rhythmical movements of the organ, and that have accumulated in consequence of there not being oxygen enough to remove them. And the reason why the muscles of the body generally show no similar change is probably that for a long time before death the patient has been resting in bed. It would be interesting to know whether the diaphragm and the intercostal muscles are or are not affected like the heart.

Muscular weakness is, indeed, one of the most marked effects of anæmia. The patient may be capable of exerting great power in a sudden effort, but he quickly becomes fatigued, and his strength is very soon exhausted. He is also incapable of undergoing mental labour, but his nervous centres are often very excitable, being, in fact, in a condition which is known as "irritable weakness." Thus there is often an exaggerated sensibility to sensory stimuli, such as a bright light or a loud noise And so, again, whereas the sexual appetite in the male is, as a rule, diminished or suspended, it occasionally happens that a morbid erethism is developed attended with frequent emissions and with inability to complete the act of coitus. In the female severe anæmia is almost always attended with amenorrhœa, and generally with a temporary or permanent sterility; but occasionally menstruation continues, and may even be profuse.

It is hardly necessary to state that the *pulse* in anæmia is small, soft, and feeble, in proportion to the severity of the case. It may even be imperceptible at the wrists. But when the cause has been a sudden loss of blood it generally has a loose, sharp, jerking character, which indicates that the arteries are imperfectly filled, not having yet adjusted their calibre to the diminished volume of the circulating fluid.

The *temperature* of the surface of the body is often low.

I have already remarked that whereas in some cases anæmia is *symptomatic*, being due to excessive losses of blood, to visceral disease of various kinds, or to exhausting discharges, other cases seem to result from a primary failure of activity on the part of the blood-forming organs. These belong in the main to two diseases, which it is convenient to distinguish as *essential anæmia;* one of them is "Chlorosis;" one is now generally known as "Pernicious Anæmia." Each must be described in some detail, and afterwards I must give an account of one of the "symptomatic" anæmiæ which resembles the "essential" forms in most of its clinical characters, but is caused by the presence of a nematode worm in the intestine.

I. *Chlorosis.*—From the time of Hippocrates this name has been applied to an affection characterised by a greenish-yellow colour of the skin, for which our English synonym has been the "green sickness." But it has now for many years been known to be merely a form of anæmia, occurring chiefly, if not exclusively, in young women. Indeed, modern writers are apt to ignore any peculiarity of complexion in this as compared with other kinds of anæmia. But I think that they are wrong, for although the special tint is not present in every case, it has seemed to me to be quite distinctive of chlorosis whenever it is present. Thus, I distrust Immermann's account, according to which it is seen only in those girls who are naturally of a dark complexion ; he says that the cheeks of brunettes, when they lose their bloom, acquire "a dirty yellowish-grey hue, contrasting with still darker greyish-blue rings round the eyes."

At one time chlorosis was supposed to be commonly caused by unrequited love. This is now known to be a mistake, but the disease may be directly excited by a sudden shock, by fright, or by some other violent emotion, as in cases cited by Trousseau. In many instances it bears a close relation to the development of the menstrual function. Thus, the age at which it shows itself is almost always between the fourteenth year and the twenty-fourth. It sometimes arises in a girl who has hitherto been robust and has had a fresh complexion, and whose breasts show no signs of immaturity, but Immermann remarks that such cases are seldom intractable. The catamenial discharge may have occurred for a certain length of time with more or less regularity, and there may be an interval after its cessation before the first signs of chlorosis are observed. Such cases have been supposed to indicate that amenorrhœa may be the cause of chlorosis, but there seems to be no doubt that this is an error. Indeed, those who become the subjects of the disease have generally from childhood been pale and delicate. Thus, Dr Ashwell in a paper in the first volume of the 'Guy's Hospital Reports' (1836), declared that chlorosis when it afterwards assumes an aggravated form has probably always existed from infancy. In many instances menstruation remains absent, and puberty can hardly be said to occur, the axillæ and the mons veneris continue to be devoid of hair, and the uterus may retain throughout adult life the form that should be peculiar to childhood. But sometimes the catamenial flow is developed prematurely. Niemeyer states that he never met with a case in which the menses appeared during the twelfth or the thirteenth year while the breasts were still undeveloped, without obstinate chlorosis following.

One can readily understand how it happens that a girl in whom the blood-forming organs have never been capable of maintaining a sufficient quantity of circulating fluid to give a healthy tint to the cheeks suffers more than others from the increased drain involved in the establishment of the menstrual function. In the same way it is easy to account for the occasional development of a condition analogous to, if not identical with, chlorosis in pale and delicate boys when their sexual organs begin to secrete the seminal fluid with its marvellous vital endowments.

Virchow has endeavoured to show that in many if not in all of those who become chlorotic at puberty there is, besides a natural deficiency of blood, a congenital hypoplasia or imperfect development of the heart and of the blood-vessels.* He finds that the aorta is much smaller and that its walls are much thinner than in healthy persons of the same age. Thus, he speaks of instances, occurring in the bodies of well-developed women, in which the aorta would hardly admit the little finger, whereas it ought to be large enough to receive the thumb; and he cites an observation, made by Rokitansky, of an abdominal aorta that was no bigger than an iliac artery should be, or even a carotid. But on referring to a brief description of some of Virchow's cases, published in 1872, I find that in several of them there was stenosis of either the mitral or the aortic orifice. Now, I have repeatedly found the aorta in its whole length extremely narrow in persons who were the subjects of obstructive disease of valves on the left side of the heart. But I believe that such affections are not congenital, but due to endocarditis occurring in childhood. Thus it seems to me that the hypoplasia of the aorta, instead of being itself a primary defect, is but a secondary result of the valvular lesion. I am not, therefore, disposed to attach much value to Virchow's observations as they stand at present. Nor does it seem to me clear that when he speaks of some chlorotic patients as being dwarfed in stature, he has been sufficiently careful to exclude the existence of acquired heart disease, which is certainly capable of interfering with the natural growth of the body.

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* "Ueber die Chlorose und Anomalien in Gefässapparate," 1872.

The *symptoms* of chlorosis are in the main identical with those of other forms of anæmia. It is worthy of notice that the patient does not grow thin; she may even become fatter than before. There is seldom, if ever, any dropsy beyond slight œdema of the ankles. I do not know that retinal hæmorrhages have been observed.

The disease is often very amenable to *treatment*, but not always. Some women have to take ferruginous remedies year after year in order to keep free from chlorosis. Indeed, those who have been the subjects of it in early womanhood are very liable to a recurrence in later life, especially if they become exhausted by child-bearing or by lactation.

Chlorosis is perhaps never directly fatal. But it sometimes seems to be the starting-point of phthisis; hysteria, chorea, simple gastric ulcer, and exophthalmic goitre are particularly apt to occur in those who are affected with it; and its presence greatly aggravates the danger of enteric fever or of any other febrile complaint.

II. *Pernicious anæmia.*—In 1855 Addison, in his work on 'Disease of the Suprarenal Capsules,' remarked that the discovery of that disease had been made by him while seeking in vain to find a cause for a remarkable form of fatal anæmia, cases of which had for a long time occasionally come under his observation. To this affection, he added, he had been accustomed in his clinical lectures to apply the term "idiopathic," by way of distinction from such anæmic states as could be traced to "the usual causes or concomitants," among which he enumerated chlorosis. Ever since it has been recognised by all of us who have been his colleagues or successors at Guy's Hospital; and allusions to it have been made in the volumes of our Reports and elsewhere.[*]

We were therefore not a little surprised to find that when Biermer, of Zürich, described it in 1868 under the name of "progressive pernicious anæmia," it was thought at first that a new disease was brought to light, and afterwards that, although Addison had recognised it some years before, it had been lost sight of until it was rediscovered by the Swiss observer. Dr Frederick Taylor collected for the volume of our Reports for 1878 no fewer than twenty-three cases which had been recorded year by year since 1854. Even before Addison's work appeared, isolated cases had been published by Coombe in 1823, by Piorry and Marshall Hall, and by Dr Barclay in the 'Medical Times' for 1851. Lebert, who was then at Zürich, wrote on it several times between 1853 and 1859.

Of all the names which have been proposed for it, I prefer *pernicious*

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[*] [*E.g.* Wilks, in the 'Guy's Reports' for 1857, and in his 'Lectures on Pathological Anatomy,' p. 159 (1859).—Ed.]

anæmia, since the epithet "idiopathic" now seems to be vague in its meaning, whereas "pernicious" is commonly accepted in Germany as a designation for fatal forms of various diseases.

Pernicious anæmia differs widely from chlorosis as regards the age and sex of those whom it affects. Among twenty-eight cases that I find to have occurred at Guy's Hospital up to the end of the year 1879, sixteen have been in males, twelve in females. Two only have been in patients under twenty, both being boys; four have occurred between twenty-one and thirty; eight between thirty-one and forty; five between forty-one and fifty; eight between fifty-one and sixty; and one at sixty-eight.

In only three of the women was the disease attributable (so far as was ascertained) to pregnancy or parturition, which have been supposed to be frequent causes for it by Lebert and by some other German writers. In no instance did it appear to have been due to a defective supply of food, as is suggested by Immermann; and it scarcely ever seemed to have been a sequel of any acute disease, or to have been preceded by diarrhœa, vomiting, or other signs of gastro-intestinal disturbance. Dr Bramwell, however, has recorded two cases of foreign sailors, in each of whom it appeared to arise out of an attack of yellow fever. And Dr Stephen Mackenzie, in a valuable lecture published in 1878 in the 'Lancet,' cites three instances in which it was caused by a severe mental shock; one patient had accidentally poisoned her father instead of giving him his medicine, another had seen a child run over in the street, and the third had been attacked by a sheep in a field, immediately before the anæmia set in.

Symptoms.—Almost always, the account which is given by a person suffering under this disease is that for some weeks or months, without any known cause, he has been getting progressively paler or weaker and more breathless. The anæmia is often extreme. But what is remarkable is that the complexion, instead of being white and waxy looking, is in many instances of a lemon or primrose-yellow tint, so that the disease has been mistaken for jaundice. The conjunctivæ, too, sometimes show a similar colour, which, I believe, mainly depends upon the presence of yellow fatty tissue beneath the mucous membrane towards each canthus. Dr Mackenzie remarks that on post-mortem examination the subcutaneous fat is often found to have an unusually bright yellow or canary colour. There may be no deficiency of adipose tissue about the body generally, but sometimes marked wasting occurs. Most patients have a bad appetite, but some eat well and even to excess. Dryness of the mouth and of the throat is often complained of; the breath has been fœtid in some instances; nausea and vomiting are frequently present, especially in the morning; and sometimes there is more or less severe pain after food; the bowels have been constipated in some cases, relaxed in others. There has always been dyspnœa on exertion, and often palpitation; one man who came under treatment at Guy's Hospital, said that every effort caused pain at the back of the head. Many patients have have epistaxis again and again; in women sanguineous vaginal discharges frequently occur; in some persons the gums bleed, or purpuric spots appear on the legs. The urine is high-coloured. The ankles commonly become more or less œdematous, and sometimes the face is very puffy. In the cases observed at Guy's Hospital there has been no considerable ascites, nor have the pericardium and the pleuræ contained any marked quantity of fluid after death. The heart always shows tabby striation.*

With the ophthalmoscope *retinal hæmorrhages* can often be detected.

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* [All these symptoms, together with the insidious origin and post-mortem appearances, were graphically described by Addison in 1855. See his collected works (New Syd. Soc.) p. 212. I have also quoted the passage in a discussion on the question of priority, which will be found in the 'Guy's Hosp. Reports' for 1882, pp. 234—240.—Ed.]

Biermer seems to have been the first to notice them. They appear either as linear striæ, or as rounded spots or patches, which may have whitish or yellowish centres, sometimes consisting (according to Manz) of accumulations of leucocytes. The optic discs are said to be swollen in some cases and prominent, their vessels to be tortuous, and the retinæ generally to have a peculiar smoky appearance. A boy, aged ten, who came under Dr Mackenzie's care, had well-marked optic neuritis. As a rule, there is no defect of vision, but Immermann says that the reason why one of his patients went to the hospital was because one eye had suddenly become blind. Niederhauser has suggested that a peculiar brittleness of the retinal blood-vessels is the cause of the greater frequency with which blood escapes from them, as compared with the vessels of other organs and tissues, but it is to be observed that after death ecchymoses of the pleuræ and pericardium are often found.

Another symptom which is, I believe, characteristic of pernicious anæmia is *fever*. It was present in all of Biermer's and Immermann's cases, and it has repeatedly been observed at Guy's Hospital. Its course is usually irregular, the thermometer sometimes rises to 104° or even higher; after a few days of high temperature there is perhaps a more or less prolonged interval of apyrexia. Why fever should occur is not very obvious, but considering how universally, in other morbid conditions, a rise of the thermometer is traceable to the entrance of some morbid material into the blood, I think that the suggestion may fairly be made that in pernicious anæmia it owes its origin to a similar cause, namely, to the absorption of some of the disintegrated matters which (as we have seen) accumulate in much larger quantities in the heart and in other structures instead of being oxidised and removed, as should be the case under normal circumstances. As a rule, there are no subjective symptoms of fever, but Immermann says that sometimes the tongue is furred, and that there may be thirst, and even shivering. In some cases the temperature rises at an early period of the disease, but usually not until it has reached an advanced stage. Before death the thermometer often falls to 97°, or even to 95°.

I have already remarked that in pernicious anæmia the red discs of the blood are very often found altered in size and in form, or with their hæmoglobin separated from their substance, but that such changes are not peculiar to this as compared with other anæmic states. It must now be mentioned that in some cases no abnormal appearances can be detected in the blood with the microscope.

Pathology.—Two theories have recently been proposed, according to each of which there is a local starting-point for the disease. Cohnheim and some other German pathologists have found that in certain cases the medulla of the bones, even when it should normally be yellow, has a deep red or purple colour, and that this is due to the presence of large numbers of cells, including many which are globular and nucleated, but which yet have a red colour. It has therefore been suggested that pernicious anæmia perhaps bears the same relation to a "myelogenous leuchæmia," as the so-called "splenic anæmia" to ordinary splenic leuchæmia. But in some instances Eichhorst and others have found the bones perfectly healthy. And, for my own part, I shall prefer to adopt Immermann's hypothesis, according to which the medullary changes, when they are present, are a result of the disease rather than the cause of it, and perhaps indicate an effort on the part of the bones to take an unusually active part in the generation of red discs, in compensation for the failure of spleen and lymph-glands to maintain the blood in a normal condition. The other theory was propounded by Dr Fenwick in the 'Lancet' for 1877. He has observed certain cases which seem undoubtedly to have been examples of pernicious anæmia, and in which the gastric glands were found after death to be atrophied to an extreme degree. He therefore thinks that this may be the primary lesion. It is an obvious objection that one would *à priori* expect wasting of the glands of the stomach to produce emaciation of the body to at least as marked an extent as anæmia. But as a matter of fact, in Dr Fenwick's cases a large amount of fat was actually present at the time of death, both beneath the skin and about the viscera. It certainly seems, therefore, that the attention of pathologists should in future be directed to this question, so that his view may either be confirmed or overthrown.

Event.—The duration of pernicious anæmia is commonly from three months to a year. In two cases which have occurred at Guy's Hospital, it is said to have run on for three years, and one patient stated that for seven and a half years he had been getting paler and weaker. According to Immermann the fatal termination is sometimes sudden, being due to syncope; but usually it is very slow and gradual, there being delirium, apathy, or even complete insensibility for two or three days before death, during which time also it has sometimes been noticed that a cadaveric odour has been exhaled from the body. As to the possibility of recovery from the disease it is difficult to speak with certainty; but when I come to discuss the treatment I shall allude to cases, believed to be genuine, which have ended favourably.

III. *Anæmia from the Anchylostomum duodenale.*—It had long been known that in Egypt a severe and even fatal form of anæmia is endemic, which indeed was commonly known as the Egyptian chlorosis. But it seems not to have been suspected that there was any special cause for the disease, which is said to attack one fourth of the population, until in 1854 Griesinger associated it with the presence of numerous nematode worms in the small intestine (*supra,* cf. p. 229). This parasite, for which another name is the Dochmius duodenalis, had originally been discovered in North Italy by Dubini; and Bilharz had already found that in Cairo scarcely any corpse that he examined was entirely free from it. Afterwards it was recognised by Wucherer in Bahia; and in 1880 it gave rise to the death of more than a hundred of the workmen who had been employed upon the St Gothard Tunnel, one of whom came under the observation of a physician formerly well known in England, Dr Bäumler, Professor at the University of Freiburg, in Breisgau.

The anchylostomum is a small round worm, with a stiff body, and a head bent nearly at right angles. The female measures about half an inch in length; the male is rather more than half as long. It may be present in enormous numbers, as many as 1250 having been counted in a single patient. It feeds, not upon the intestinal juices, like other worms, but upon blood, which fills its digestive canal, and gives its body a red colour. It fixes itself firmly by means of three pairs of hooks into the substance of the mucous membrane of the duodenum or of the jejunum; and within its mouth there are two moveable cutting blades which doubtless serve to incise the tissues still further. The spot to which it is attached is indicated by an ecchymosis; and Leuckart thinks that it shifts its position from time to time, and that the punctures which it leaves may then go on bleeding into the cavity of the bowel, which is sometimes found filled with blood after the patient's death, although hæmorrhage per anum seldom, if ever, occurs during life. The body of the worm commonly hangs free within the gut, protected more or less by the ridges of the mucous membrane; but sometimes it is rolled up in a hollow space in immediate contact with the muscular coat. The female anchylostomum throws off numerous eggs, which are oval bodies, with a thin transparent shell, of nearly the same size as those of the oxyuris, but less elongated, measuring only $\frac{1}{20}$ mm. in length. They also differ in having no operculum, and in having their yolk undivided or simply segmented at the time of their expulsion in the fæces, whereas the eggs of the oxyuris already contain embryos. The life-history of the anchylostomum has not been directly traced, but it is believed to be the same as that of the allied *Dochmius trigonocephalus* of the dog. If this be correct, the ova become hatched when they pass into mud or water, and produce slender worms which exhibit very active movements. These require no intermediate host, but develop into sexually mature animals when they are swallowed and reach the human alimentary canal. It is not surprising that the parasite should be met with chiefly in hot climates, where men are often compelled to drink water from dirty pools exposed to contamination in every way. The resulting anæmia may prove fatal in a few weeks, or it may run on for years until death perhaps occurs by dysentery or by some intercurrent acute disease. At first the patient appears well nourished and even fat, but at length he becomes wasted and dropsical. The only special symptoms which I find mentioned are disorders of digestion and a cutting pain in the abdomen.

The *diagnosis* of the different forms of anæmia sometimes involves questions of great importance and of much difficulty. Experience shows that there is no little danger of mistaking it for jaundice. This is especially apt to be the case with pernicious anæmia. In 1877, a man, aged thirty-eight, was in the clinical ward of Guy's Hospital for seventeen days with what was believed to be either acute yellow atrophy of the liver or cirrhosis; he became delirious and violent, and had to be placed in a separate room; afterwards he was insensible, but his alarming symptoms ultimately subsided and he was discharged. However, three months later he returned to the hospital, and it was then obvious that his disease was merely anæmia. He sank and died, and the liver was found to be healthy. I have seen several instances of the same kind, and scarcely less striking. So again, it often happens that the anæmia of patients suffering from cancer is supposed to be jaundice, and is taken as proof that the liver is secondarily affected. As I have already remarked, the appearance of the conjunctivæ may afford little if any help in distinguishing the two conditions, since in pernicious anæmia the eyes often have a distinctly yellow tint. But I had always thought that the state of the urine was conclusive, until I read in the report of the case just alluded to that traces of bile were discovered in the urine when the patient was in the hospital for the first time. And even now I am inclined to think that this statement may have been erroneous.

When the morbid state has been recognised as anæmia, the next point to be considered is whether hæmorrhage can have been the cause of it. It must not be imagined that to determine this is always easy. From motives of delicacy persons will sometimes conceal the fact that there has been draining from hæmorrhoids, or from the uterus. Indeed, serious bleeding may occur per anum without the patient's being aware of it. In 1880 I was called to see, with Mr Earle of Brentwood, a young lady who had fallen into a state of the most alarming prostration and bloodlessness. She had no doubt been ailing for some time, but she had been able to play lawn tennis until two days before my visit. She positively assured us that she had noticed no hæmorrhage from any of the mucous surfaces, and some days passed before her mother made the discovery that the evacuations from the bowel contained blood. Ultimately an ulcer was detected in the rectum. I am inclined to think that the suddenness of the girl's illness would almost have justified one in asserting that there must be such a cause for it.

One can doubtless classify either under chlorosis, or else under the pernicious form of the disease, a large majority of the cases in which a well-marked anæmia is "essential." But it would be a great mistake to give a bad prognosis whenever the patient has reached the middle period of life. I have before me notes of the case of a lady, aged forty, who came to me on January 7th, 1881, in a state of extreme bloodlessness, which had been coming on for eleven months. I was quite under the impression that the disease would prove intractable, but on January 26th she returned with lips and hands fairly coloured, with a good appetite, with no sickness (whereas she had vomited every morning), and with no dyspnœa (whereas to walk upstairs had caused her great distress). As she told me, "colour appeared on her cheeks in small spots on the fourth day" after she began to take the tincture of iron which I had prescribed for her.

On the other hand, it is very important to remember that one may mistake for chlorosis—or it would perhaps be more correct to say, that chlorosis may mask the development of—serious, and even fatal, organic disease. In speaking of *gastric ulcer,* I had to mention that many girls who seem to be simply chlorotic are attacked with fatal peritonitis from perforation of an ulcer which had given rise to no symptoms. A point of even still more importance is that we should be on our guard against the insidious onset of tuberculosis. The early stages of pulmonary *phthisis* are often overlooked in patients whose complaints are attributed to anæmia, and the only way to avoid such mistakes is to have the evening temperature taken at frequent intervals, and to examine the upper lobes of the lungs from time to time with the stethoscope.

In 1861 a girl, aged eighteen, who was admitted into Guy's Hospital for chlorosis, gradually sank and died; a large scrofulous mass, which seems to have produced no marked symptoms, was found in the cerebellum, and there were a few scattered tubercles in the lungs. I have also seen cases of peritoneal tuberculosis which have been scarcely less obscure.

Again, in 1866, a man, aged sixty-seven, lay for a long time in the clinical ward with what was believed to be the "idiopathic anæmia" of Addison, but at the autopsy it was found that the lungs were full of miliary tubercles, and that the liver, the spleen, and the kidneys also contained them in smaller numbers. Of far less practical importance, but still worthy of attention, is the fact that one may sometimes mistake for pernicious anæmia other incurable diseases. In 1879 I made an autopsy in the case of a woman, aged fifty-nine, whose skin had been of a lemon-yellow colour, and whose blood had been found to contain red discs of irregular form, so that the diagnosis had been supposed to be beyond question. However, the heart weighed twenty ounces, the minute arteries in the pia mater were greatly thickened, and the kidneys, although large, were hard and glistening, and showed an excess of fibrous tissue under the microscope. It was a case of chronic Bright's disease. In other instances a condition of anæmia, supposed during life to have been "essential" and "pernicious" has been shown in the post-mortem room to be the result of the development of *cancer* in the stomach, or in some other organ, without any local symptoms having resulted from it.

When there is a doubt as to the possible presence of the *anchylostomum* in a case of anæmia, it is important to examine the fæces of the patient with a microscope, since the ova of the parasite can be recognised in them without difficulty.

In regard to the *prognosis* to be given in cases of anæmia I have little to add to what I have already stated in describing its several forms, but when hæmorrhage is the cause it is important to remember that infants and very old persons are apt to succumb under the effects of a loss of blood which would at other periods of life be attended with but little danger.

In cases of essential anæmia one does wisely in giving no forecast until the patient has been a little while under *treatment.* For the effects of the preparations of iron often appear quite marvellous, especially when they are watched day by day by means of the hæmacytometer. Thus, in a case of chlorosis recorded by Dr Gowers in the 'Lancet' for 1878 the red discs were at first found to be reduced to 26 per cent. of the normal (1,290,000 per cubic millimetre). Iron was administered, and at the end of a week their numbers rose to 40 per cent. (2,000,000 per cubic millimetre), while twenty-six days later they had reached 76 per cent. (3,800,000 per cubic millimetre). The fact that the metal is so important a constituent of hæmoglobin suggests that it should perhaps be regarded as a food rather than as a medicine; but to make this view tenable one would have to assume that there has been a deficiency of iron in the dietary of those who are benefited by it; and it is certain that such is not always, nor even generally the case. One can therefore only suppose that it stimulates the process by which red discs are developed.

In some cases in which there is much stomach disorder it is advisable to give only bland preparations (such as the ammonio-citrate or the potassio-tartrate) or even the tasteless solution of dialysed iron. But it often happens that the tincture of the perchloride, or the sulphate, can be borne by patients who have a pale flabby tongue, nausea, vomiting, and even pain after food; and such treatment is generally attended with signal success. Large doses seem to be much more serviceable than small. Probably their advantage lies mainly in the fact that but a very small part of what is swallowed is absorbed into the blood, it having first to enter into combination with an albuminous principle; all the rest is converted into a sulphide in the small intestine, and passes away in the fæces, giving them the well-known black colour.

But in pernicious anæmia the preparations of iron have unhappily proved to be altogether ineffectual. Too often no medicine can be found which will check its downward progress. Arsenic, however, has sometimes been very successful in cases which appeared to belong to this disease. Dr Bramwell has recorded two such instances ('Edin. Med. Journ.,' Nov., 1877); one is that of a man, aged thirty-eight, admitted for extreme anæmia, with retinal hæmorrhages, who had been ill seven months, and who became much worse under the administration of the tincture of iron which was continued for a fortnight, but who completely recovered in the course of four months, while he was taking the liquor arsenicalis in doses gradually increased from two to twelve minims. Phosphorus has sometimes been thought to be useful. Dr Pye-Smith had under his care in Guy's Hospital a patient who regained his appetite for a short time when one twentieth of a grain of phosphorus was prescribed for him, and became able to get down into the hospital garden; however, he afterwards relapsed and died.*

It very seldom happens that the tranfusion of blood can be employed with advantage in cases of anæmia that come under the care of the physician; but this procedure must never be forgotten when a patient seems to be in danger of sinking, and when the results of previous treatment have not already shown that he is suffering from incurable disease. In pernicious anæmia it has been employed by Quincke and by others; but seldom, if ever, with any good result, even for a time. Human blood only should be employed, and it should be defibrinated, by stirring, before it is injected into the patient's circulation, on account of the danger of fatal embolism when it is allowed to retain its power of coagulating. That the red discs remain as living elements of the blood, after having been transferred from one person to another, seems to be certain from the fact that no change in the urine is observed after the operation, whereas when lamb's blood is used, except in very small quantities, an invariable result seems to be the escape, by the kidneys, of hæmoglobin which has been set free by the disintegration of the heterologous blood-corpuscles. A sense of oppression, or of suffocative distress, is sometimes complained of while the operation is in progress; but this may to a great extent be obviated if the syringe is used slowly and gently. The largest quantity that should be injected at once is half a pint; in many cases from four to six ounces suffice.

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* [In the 'Guy's Hospital Reports' (vol. xlii) I have collected 129 authentic cases of Addison's anæmia, and of these no less than 29 have ended in apparent recovery; so little does it deserve the epithets "progressive" and "pernicious." The case there given at p. 277, which recovered under arsenic after retinal hæmorrhage, has recently come again under my notice. The patient is now (July, 1885) perfectly well. To the list of cases and authors at the end of that paper I now add an historical notice by Dr Musser in the Philadelphia 'Med. News,' Oct. 7, 1882, and a treatise on 'Anæmia,' with numerous temperature charts, by Dr Laache, of Christiania, 1883.—Ed.]

The *Anchylostomum duodenale* may be effectually removed by the ordinary anthelmintics, such as santonin or the oil of male fern.

HÆMOPHILIA

History—Inheritance usually in males through females—Course and symptoms —Prognosis—Diagnosis—Pathology—Treatment.

From the commencement of the present century it has been known that in certain families the males during successive generations are liable to protracted and even fatal hæmorrhage after injuries of no great severity, or, as it is termed, are "bleeders." Attention was pointedly drawn to the subject in 1803 by an American physician, Otto, who gave an account of a family in which this morbid tendency had existed for seventy or eighty years ('Philadelphia Med. Repository'). In the older medical literature a few allusions to it have since been discovered, the earliest being in the writings of an Arabic author who is said to have died at Cordova in A.D. 1107. In 1784 Sir William Fordyce recorded the case of a Northamptonshire family affected in a similar way. During the last sixty years German writers chiefly have dwelt upon this morbid condition, to which Schönlein gave the barbarous name of hæmorrhaphilia, since abbreviated into Hæmophilia. The latter designation is now universally adopted, having replaced in this country that of "the hæmorrhagic diathesis," which was at one time in vogue. The most important treatise on it in the English language was published by Dr Wickham Legg in 1872 (see also 'Path. Trans.,' 1885, p. 416).

Heredity.—We have seen that the hereditary transmission of hæmophilia is one of its most remarkable features. Its isolated occurrence is, indeed, exceedingly rare; a collection of all the known cases is said by Immermann to give an average of three to each family. When a "bleeder" is born of healthy parents, it almost always happens that some or all of their subsequent children are also affected; and we may reasonably suspect that the disease had already existed in a grandparent or some ancestor. The preponderance of males among those who suffer from hæmophilia is enormous, being about as thirteen to one. Moreover, hæmophilia is much less severe in women, and is scarcely ever fatal to them, the signs of it being often merely a tendency to the occurrence of cutaneous ecchymoses, spontaneously or after slight injuries—or, in other cases, to epistaxis, to menorrhagia, or to excessive post-partum hæmorrhage. But Immermann (in Ziemssen's 'Handbuch') says that an instance has been recorded by Reinert, in which, among a mixed family of sons and daughters, the sons were all free, the daughters alone being attacked.

It is therefore a very striking circumstance that the inheritance of the complaint takes place mainly through the female line. The sons in a family in which it exists do not always all suffer; and if any escape their children are almost invariably exempt. Even those sons who are affected, if they live to beget offspring, not seldom have some boys who are healthy. But the daughters of such a family, even when they have not themselves shown the slightest indication of hæmophilia, are almost certain to transmit it to their male children. Obviously these facts are very important in regard to the advice which should be given if one is consulted as to the propriety of marriage on the part of different members of a hæmophilic race. At Tenna, in the Grisons, there were until lately two families, not known to be related to one another, in which the disease had been known to exist for a century (Vieli). In 1855 the females of these families determined to renounce marriage for themselves, and in 1879 Immermann was able to state, on the authority of Dr Hörsli, of Thusis, that there was no longer a well-marked example of hæmophilia in the village. A curious circumstance is that persons with an inherited tendency to the disease seem to be unusually prolific, the average proportion of children to each union being nine, whereas among the population generally it is five. No especial type of bodily configuration seems to be associated with hæmophilia. It occurs in those who are lean as well as in those who are fat, and in persons whose hair and eyes are of all varieties of colour. Those who are attacked by it are, however, said to have generally a thin, delicate, and transparent skin, with obvious and full subcutaneous veins. The disease has repeatedly been observed in European Jews, and if a large proportion of recorded cases have occurred in nations of Teutonic as compared with those of Latin origin the reason probably is that it is only in certain countries that much attention has yet been given to the subject.

Course and symptoms.—Hæmophilia does not usually appear at the time of birth. The detachment of the navel-string has but rarely been attended with bleeding in children who were afterwards to suffer from it. But about the end of the first year, or at least before the close of the second year, definite symptoms generally manifest themselves. The latest age at which it is said to have ever commenced, when it was to assume a serious form, is during the twenty-second year. The hæmorrhages which characterise it are commonly divided into those which are traumatic and those which are spontaneous, but the distinction is only partially applicable, since slight injuries are very apt to be forgotten which in hæmophilic subjects may give rise to considerable extravasations. Thus spots and patches of effused blood in and beneath the skin may sometimes be traced to the pressure of the clothes. But there can be no doubt that they sometimes arise independently of any such cause, especially where they are small and such as would be termed petechiæ. Indeed, successive crops of a cutaneous purpura may be observed in this disease exactly as in the "morbus maculosus" and in so many other morbid states. The spontaneity of hæmorrhages from mucous surfaces is said to be sometimes shown by their being preceded by well-marked signs of "fluxion"—throbbing of the heart and of the arteries, redness and heat of the cheeks, ears, and lips, headache, giddiness, irritability of sight and hearing, restlessness, &c. Epistaxis is the most common form of mucous hæmorrhage, especially in children. According to Grandidier it is four times as frequent as any other. Next in order of occurrence comes bleeding from the gums and mouth. This, however, may be traumatic. Dr Legg mentions that some patients cannot use an ordinary tooth-brush without drawing blood; or there may be hæmorrhage from the stomach, the intestines, the lungs, the urinary passages, the female genitalia, or even the lachrymal caruncle. Blood does not often escape from an unbroken surface of the skin, but cases have been recorded in which it has oozed from the finger-tips, the ears, or even the back of the head.

In marked contrast with these spontaneous or quasi-spontaneous forms of hæmorrhage are those which result directly from blows or cuts. Even slight superficial scratches, such as would scarcely be noticed in a healthy subject, may bleed so as to endanger life. There are, however, considerable differences as to the amount of injury that can be borne, not only in different hæmophilic patients, but in the same patient at different periods. At one time a cut may cause but slight loss of blood, whereas there may be the greatest difficulty in checking the oozing from a precisely similar one on a later occasion. The operation of ritual circumcision has several times proved fatal. So have venesection, the application of leeches or cupping glasses, and, above all, the extraction of a tooth. Indeed, although Dr Legg says he has seen a tooth drawn without there being any remarkable amount of hæmorrhage, both he and all other writers are agreed that it should scarcely ever be done, for the relief of toothache, in those who are "bleeders." The slight punctures which have to be made for vaccination have scarcely ever given rise to much bleeding. It is to be observed that the risk of hæmorrhage from a wound continues until it is completely healed; a thin cicatrix has even been known to give way afterwards.

The deeper structures may also be the seat of extravasations of blood, which reach an enormous size, and which are somewhat peculiar to hæmophilia, in contrast with other diseases of the same order. Thus a subcutaneous tumour may be formed, as large as an apple or even as a child's head. It sometimes seems to arise spontaneously, but very often it is due to some slight injury. Thus, in a case of Sir William Jenner's, cited by Dr Legg, the fall of an india-rubber air-ball upon the thigh filled the connective tissue with blood from the knee to the trochanters. The thigh is, indeed, one of the favourite seats of such swellings, and Immermann says that they are also frequently seen over the false ribs, and upon the back. They are generally of a black or dark-blue colour, surrounded by a zone of red. They are sometimes very hard, sometimes soft and even fluctuating. They may be hot, and painful and tender to the touch; and in some cases they suppurate, discharging a mass of altered blood with shreds of broken-down tissue, after which hæmorrhage from their floor is apt to go on for a considerable time. If they are punctured by the surgeon, dangerous bleeding commonly follows. As a rule, they slowly subside and at last disappear.

In hæmophilia the effusion of blood into any one of the large serous cavities seems to be rare. Immermann (in Ziemssen's 'Handbuch') cites two cases in which the peritoneum was the seat of hæmorrhage, and four in which it occurred into the cerebral membranes; but in at least three of these latter there had been a fall or a blow upon the head. He says that extravasations into the pleural sac or into the pericardium have not yet been observed. It may therefore be worth while to note that in a case in which Dr Goodhart made an autopsy the right pleura was closed by adhesions which were in part stained of a deep orange colour.

Articular affection.—One of the most remarkable features of the disease has still to be mentioned, namely, the occurrence of swelling in one or more joints, especially the knees. This affection usually begins between the seventh and the fourteenth year. It is sometimes the direct result of a blow, and sometimes it immediately follows a long walk; but Dr Legg says that the most common cause is exposure to cold or the occurrence of cold damp weather, and that it is most frequently seen at the beginning of spring and at the end of autumn. The enlargement often occurs rapidly, and appears precisely like that which might be due to rheumatism, or to synovitis. Many writers have therefore expressed the opinion that it often depends upon simple serous effusion into the articular cavity. But all the pathological evidence which at present exists seems to point to the conclusion that the primary change is an extravasation of blood. It is true that when a joint has been affected for a length of time the cartilages show signs of chronic inflammation, and that the projecting folds of synovial membrane in its interior are thickened and swollen. Mr Symonds tells me that this was markedly the case in the knee of a boy who was under Mr Bryant's care at Guy's Hospital in 1880, and who had had the part more or less enlarged for three years. But in the same patient there were other joints in which at the post-mortem examination all the strictures were healthy, except that they were stained by orange-coloured pigment and had stringy masses of ochry-brown fibrin adherent to them. It might perhaps be supposed that if a great quantity of blood were poured out into a knee-joint, a discolouration must be visible through the skin, but I think that this is negatived by observations which I have made in gout. The usual course of the articular affections of hæmophilia is slowly to subside under treatment, but to return again and again at intervals of months or years.

Prognosis.—In some very exceptional cases of hæmophilia the hæmorrhagic tendency is said to cease during childhood or youth, and the patient afterwards remains free from the disease. But by far the larger number of those who are affected with it die before they are eight years old. When adult life has been reached the danger is consequently lessened, but it is by no means at an end, for a fatal bleeding has been known to occur as late as fifty or sixty years of age. The habitual condition of hæmophilic persons, even when they have suffered from the disease for a length of time and are perhaps still troubled with a joint-affection, is not generally one of permanent bloodlessness; they often have as much colour in the face and lips as people in general. But the immediate effect of a profuse loss of blood is of course an extreme degreee of anæmia. The quantity poured out is sometimes enormous. One case is related in which, after the extraction of a tooth, half a gallon was lost in less than twenty-four hours. It often happens that oozing goes on at the rate of three or four pints a day for several days. When the source of the hæmorrhage is visible, it seems to come, not from a single vessel, but from a surface, as of a saturated sponge. Sir William Jenner has remarked that it has generally appeared to him to be venous rather than arterial. As the bleeding goes on, the patient becomes blanched, pulseless, delirious and unconscious, and death is often preceded by convulsions. But sometimes, when his vital powers are reduced to the lowest ebb, the oozing, which may have resisted all treatment, ceases spontaneously; he remains apparently on the brink of dissolution for several days, and then slowly revives. Writers generally say that the circulating fluid undergoes restitution in such cases more rapidly than might be expected, but Dr Legg remarks that the anæmia at any rate remains for four or even six months.

Diagnosis.—The diagnosis of hæmophilia is not difficult in confirmed cases. But one must keep in mind the possibility that a joint-affection may be due to this cause, even when it occurs in a person who may not be anæmic, who may exhibit no purpuric spots, and who may not think of mentioning that he is a bleeder. The real nature of less marked examples of the disease, such as occur in the female sex, could probably never be positively determined without reference to the history of family predisposition. Many women have a tendency to bruise very readily, and others are subject to the recurrence of spontaneous hæmorrhages, especially in the form which has attracted so much notice under the name of "hæmatidrosis," and in which blood oozes from the mouth of the hair-sacs or sweat-glands of various parts of the skin. In the 'Med. Times and Gazette' for 1871, Dr Legg has recorded two cases, in women, of a "hæmorrhagic diathesis," in which the resemblance to hæmophilia was nearly complete, there being a great liability to hæmorrhage from slight wounds, and also to epistaxis, menorrhagia, and purpura. In each instance the abnormal state had been present for some years, but in neither of them did it exist before puberty. This last circumstance, however, could hardly have been held to exclude hæmophilia, because many of the slight forms which are seen in women seem not to be recognisable during childhood. But what appeared to be conclusive was that each patient had borne male children who were not bleeders, and that no family history of hæmophilia could be elicited. On the other hand, the fact that there had not been any antecedent tendency to hæmorrhage seems to be a good ground for rejecting certain cases which have been classed with hæmophilia by writers; as, for instance, when the only thing in favour of such a diagnosis has been that the patient bled to death after what appeared to be a not very severe injury.

Pathology.—With regard to the nature of hæmophilia very little can as yet be said. Some of the older writers supposed that it depended upon a defective coagulability of the blood. But it is now known that this was a mistake, and there is no proof whatever of any abnormal condition of the circulating fluid, except as the result of the continuance of hæmorrhage. So, again, the fatty change in the substance of the heart which has sometimes been found is clearly consecutive to the anæmia which commonly precedes death. Several observers, however, have noticed a peculiar thinness and transparency of the arteries—not only of the aorta and pulmonary artery, but also of such branches as the temporals and radials. Others, indeed, have failed to discover anything of the kind. And the most that could be said about it is, I think, that, although not itself the cause of the symptoms of the disease, it may possibly point to the existence of a similar abnormality of the capillaries, and that this may really be the cause. Thus Dr Legg suggests that hæmophilia may depend upon a backwardness of growth or imperfect development of the vascular system generally. Immermann also speaks of a "hypoplasia" of the vessels, but he thinks that the essential thing is a disproportion between the capacity of the circulatory apparatus and the volume of the blood. He imagines that in hæmophilic males there is generally an actual overgrowth of the blood itself, and he endeavours to account for the transmission of the disease by females who themselves are not bleeders by supposing that they have the thin vessels, without blood excess. Sir William Jenner's authority may be quoted ('Brit. Med. Journ.,' 1876, ii) for the view that there is in hæmophilia "a tendency to plethora of the smaller vessels." He remarks that it is when the patient is looking his best that injuries have the worst effect, and that spontaneous hæmorrhages are the most likely to occur.*

Treatment.—These views are not without bearing upon the management of hæmophilic subjects. The diet should be light and stimulating. Jenner recommends "a considerable proportion of white meats." An aperient dose of sulphate of soda may be given every week, and a mercurial every third week. On the other hand, Dr Legg has found that after taking the tincture of perchloride of iron patients have been less liable to spontaneous bleedings and have had but little loss of blood after wounds. A warm, dry climate is desirable. When actual hæmorrhage is going on, the tincture of iron, or ergot, or gallic acid may be used internally. Locally there appears to be no styptic so valuable as the perchloride of iron; a strong solution may, if necessary, be applied to the interior of the rectum; when the socket of a tooth bleeds after extraction solid crystals of the salt may be introduced into it.

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* [In a paper by Dr Percy Kidd ('Med.-Chir. Trans.,' vol. lxi, p. 243) he describes changes in the minute vessels.—Ed.]

PURPURA

Symptomatic and primary purpura—Ætiology—Characters, events, and treatment—Morbid anatomy—Pathology—Diagnosis.

I have repeatedly had occasion to mention under the name of *Purpura* the formation of spots of hæmorrhage in and beneath the skin. It occurs, for example:

1. In scorbutus. 2. In hæmophilia. 3. With rapidly diffused and infective forms of sarcoma. 4. In Hodgkin's disease. 5. In splenic leuchæmia. 6. In pernicious anæmia. 7. With chronic lesions of the liver or of the kidneys. 8. In ulcerative endocarditis. 9. As a consequence of certain cutaneous affections, especially "erythema multiforme," and the exanthem which precedes the usual eruption of smallpox.

In all these varieties the cutaneous hæmorrhages are secondary, nor are they by any means universally present. But there is another form of purpura of which they constitute the fundamental and the essential symptom, and for which at present no cause can, as a rule, be discovered. In Germany this disease is commonly spoken of as the "Morbus maculosus Werlhofii." It was, in fact, described in the last century by Werlhof, a physician whose name may not be without interest to Englishmen, since he held a Court appointment to the King of Great Britain in Hanover; he died in 1767. In his 'Opera Medica,' collected by Wichmann, a well-marked case is recorded; the patient was a girl previously healthy, who also had epistaxis, hæmatemesis, attacks of syncope, and a small and very rapid pulse; she ultimately recovered.

Of course it cannot be pretended that Werlhof would have exactly limited his morbus maculosus to what we now regard as an essential purpura. Nor have we ourselves any certain warrant for assuming that this may not hereafter be still further broken up into a series of separate affections, owing their origin to different causes. There is, indeed, a subdivision already of some antiquity, and accepted by most writers, into a "P. simplex" (confined to the skin) and a "P. hæmorrhagica" (attended with bleeding from various mucous membranes and with ecchymosis of the deeper structures of the body). That, however, is altogether artificial and unnecessary, the distinction being merely one of degree. For the less severe cases of purpura alone belong to the so-called "simple" form of the disease, and they are very apt after a few days to pass into the "hæmorrhagic" form.

Ætiology.—The morbus maculosus seems to be more common about the age of puberty than at any other period of life; females are said to be more subject to it than males.

The patient is often well nourished and fresh coloured, having had good food and having been apparently quite well up to the time when the cutaneous spots are observed, or when hæmorrhage begins from some mucous surface. Sometimes he is anæmic and weakly; or he may have recently recovered from an acute disease, such as enteric fever. Immermann (in Ziemssen's 'Handbuch') remarks that the purpura then usually shows itself first when the appetite is returning and when the first attempts to stand are being made. Now and then, in cases in which there has been no antecedent malady, there is a short prodromal stage of malaise, annorexia, and headache, lasting two or three days or even a week.

But in some cases the occurrence of purpura is directly attributable to the medicinal administration of the iodide of potassium, or of ammonium, the corresponding sodium-salt being generally incapable of producing a similar effect. This "iodic purpura" appears to be in all respects identical with the ordinary form of the disease. For descriptions of it, with copious references, I may point to papers by Dr Stephen Mackenzie (in the 'Med. Times and Gaz.' for 1879) and by Dr Duffy (in the 'Dublin Med. Jour.' for 1880). In one case—that of a young syphilitic infant, five months old —it is said to have directly followed a single dose of two and a half grains of iodide of potassium; but it is generally not seen until the salt has been taken for some days or even for several weeks. The spots may sometimes subside, notwithstanding that the patient goes on with his medicine; but in most cases, should he have discontinued it, a fresh crop of them appears as soon as he attempts to resume it.

Symptoms.—Not infrequently the legs are alone affected by purpura. Or the spots may appear on them earlier than anywhere else, the forearms being the next parts to be attacked, and the face suffering last, or not at all. But sometimes they come out simultaneously over nearly the whole of the cutaneous surface. They may be in untold numbers. They present differences of colour, which chiefly depend (as Dr Hyde Salter pointed out in the 'Med. Times and Gazette' for 1856) on the depth in the skin at which the blood is extravasated. Thus the more superficial of them, which are seen through only a thin layer of tissue, appear bright red and sharply defined; the deeper ones are of a purple hue and fade off gradually at their edges; they are also generally larger, because the meshes of the tissue in which they lie are more open. Very rarely the cuticle is raised into a dark bleb by blood poured out upon the surface of the rete. The spots bear no definite relation to the hair-sacs, such as has been described in scorbutus. Nor is there so marked a tendency to the formation of large subcutaneous vibices, but the eyelids are sometimes surrounded by broad black rings, and extensive effusions of blood may sometimes be seen in other regions, especially when there have been blows or injuries.

Among the mucous membranes, that of the nose is perhaps more apt than any other to bleed in the "morbus maculosus;" epistaxis is not seldom the earliest symptom. Hæmorrhages also frequently occur from the stomach, the intestines, the urinary passages, the female genital organs; and sometimes, I believe, from the bronchial tubes. Blood may ooze from the gums, and collect round the bases of the teeth in dark red or black crusts. But when these crusts are removed, the gingival tissues are never found to be swollen, spongy, nor of a purple-red colour, as in scorbutus; they are either perfectly normal in appearance, or more or less pale and anæmic.

In the more severe cases of purpura, when there has been a considerable loss of blood, the patient may rapidly pass into a condition of extreme anæmia, with waxy pallor of the skin, a rapid feeble pulse, and a liability to faint on the slightest exertion, or even on attempting to sit up. Under such circumstances fever may be present, as in all other forms of anæmia. Immermann further suggests that the temperature may sometimes rise as the result of the reabsorption of the extravasated blood, or in consequence of the setting up of local inflammatory changes by its irritant action upon the tissues among which it lies. It seems still to be doubtful whether fever is ever present as an initial symptom, or at least whether the occurrence of a high temperature at the beginning of an attack of purpura does not show that the case is of a peculiar kind. With regard to the condition of the blood within the vessels in the morbus maculosus there have been many discrepancies of statement. It has been said to be deficient in coagulating power, but this seems to be a mistake. Immermann had an opportunity of estimating the proportion of leucocytes to red discs in a very severe case which occurred at Basle; during the first few days it was normal, but afterwards there was a slight excess of leucocytes, as is usual after all forms of hæmorrhage.

Event.—As a rule, purpura ends rather quickly in recovery. Even in some cases fresh spots may cease to come out after a few days, the old ones fade and disappear, the mucous membranes cease to bleed, the anæmia is quickly repaired, and within three or four weeks the patient is as well as ever. If he should get up too early it often happens that a new crop of spots may be seen upon his legs within an hour or two of the time when the feet are first allowed to touch the ground; but these generally soon undergo absorption in their turn. Sometimes, however, the disease runs on for several weeks, or it may recur again and again, with intervals of many months, during which the health appears to be perfectly good.

Treatment.—There is reason to believe that certain medicines are capable of preventing the formation of fresh spots of purpura and of averting the hæmorrhages from mucous membranes which constitute the most serious part of the disease. Of these I believe that *arsenic* is the most valuable. Dr Habershon recommended it in the 'Guy's Hospital Reports' for 1857; it has ever since been commonly employed at the hospital, and I had one case in which it succeeded at once, when many other drugs had failed. Turpentine, again, seems to be useful in some instances; and ergot, acetate of lead, dilute sulphuric acid, may each be prescribed in turn. Immermann says that it is important not to begin to treat the consecutive anæmia by ferruginous preparations until some time has passed, and that their administration has sometimes led obviously to a relapse of the disease. The patient should be kept in bed in a cool room; he should have a light milk diet, his bowels should be relieved by gentle laxatives.

Fatal cases.—But sometimes the morbus maculosus proves fatal. The patient may sink, exhausted by repeated hæmorrhages from mucous surfaces, after two or three weeks' illness. The linings of the stomach, intestine, uterus, kidneys, and bladder are then generally found spotted with ecchymoses, as are also the pleura, the pericardium, the arachnoid, the peritoneum, and even the substance of the lungs and the medulla of the bones. In some cases an effusion of blood upon the surface of the brain or into the ventricles is the direct cause of death. Thus a man, aged thirty-four, was admitted into Guy's Hospital for purpura, and appeared to be doing well, when he became insensible and paralysed on the right side. Afterwards there was loss of power in the left limbs also, and he died on about the twentieth day of his illness. A quantity of blood was found extravasated on the left hemisphere of the brain beneath the pia mater, and also within the ventricular cavities. Another case, which occurred in 1871, was that of a woman, aged twenty, who, while in the hospital for phthisis, was attacked with a severe form of purpura, and with epistaxis. At the end of about a week she became delirious for some hours on two successive days. However, the bleeding ceased, and the spots disappeared, and she sank gradually a fortnight later with diarrhœa. The dura mater over each hemisphere was then found lined with a uniform layer of blood, which was yellowish in tint and had assumed a membranous character. In other instances, again, some of the more delicate structures of the body seem to slough as the result of the infiltration of blood into their tissues. Thus a girl, aged nine, was admitted into Guy's Hospital for gangrene of the external genitalia and purpura. So far as could be learnt she had not been affected with any one of the contagious exanthemata. There was a fœtid discharge, and she died in four or five days. The bladder, the vagina, the uterus and the Fallopian tubes were all found intensely inflamed and covered with spots of hæmorrhage. Some years later a man, aged twenty-three, died after an illness of eight days' duration, which began with a purpuric affection of the right thigh. The lower end of the ileum, for about one foot of its length, was of a purple colour, and had its coats thickened and infiltrated with exudation, its serous surface coated with lymph, and its mucous surface slightly excoriated. Zimmermann has even related in the 'Arch. f. Heilkunde' for 1874 a case in which several intestinal ulcers formed and sloughed through into the peritoneum, setting up a fatal peritonitis.

Pathology.—I have purposely left it to the last to discuss the pathology of the morbus maculosus; and, indeed, we have very little accurate knowledge with regard to it. There are good analogies in support of the view that it depends primarily upon a peculiar dyscrasia or alteration in the constitution of the blood; but it seems clear that before the hæmorrhages occur, this must have led to a morbid state of the walls of the capillaries, perhaps by impairing their nutrition. Immermann suggests that the purpura which occurs during convalescence from fever is possibly due to the circumstance that the recovery of the tone of the minute vessels is sometimes retarded beyond the time at which the volume of the blood is restored and the heart regains its vigour. In an isolated case, recorded by Dr Wilson Fox, in the 'Med.-Chir. Rev.' for 1865, the arterioles and capillaries of the skin in the neighbourhood of purpuric patches were found to be obviously altered in appearance; they were brittle, had a glistening waxy look, and assumed a most intense reddish-brown colour with iodine. The patient, a man aged thirty-three, had been affected for about a month with a syphilitic eruption which followed an indurated chancre at five months' interval. He had taken iodide of potassium for some time, but not continuously. It seems doubtful whether any of the viscera were lardaceous, except perhaps the adrenal bodies and the intestinal mucous membrane. Many of the muscles were in parts pale and waxy looking; their fibres had lost their striation and become deeply stained by iodine, while their blood-vessels showed changes similar to those observed in the affected parts of the skin. Dr Thin has recently ('Med.-Chir. Trans.,' lxii) described the minute blood-vessels as obviously "altered," and "disorganized," within the area of a bulla caused by the internal administration of iodide of potassium; and he suggests that iodic purpura is due to a still more extreme change in them.*

Diagnosis.—The diagnosis of the "morbus maculosus" rests upon the exclusion of all the various diseases which may give rise to a "symptomatic" purpura. The possibility of the sporadic occurrence of scorbutus must not be forgotten, but even mild cases of it are generally distinguished by several positive characters, of which the chief are the debility and anæmia that precede the cutaneous affection, the swollen and spongy state of the gums, the brawny induration in the hams, the formation of the spots each round the mouth of a hair-sac. On the other hand, in some cases of purpura, hæmorrhages from the mucous surfaces are generally much more profuse than they are in scorbutus. Of hæmophilia all that need be said is that its special characters have sometimes to be elicited from the patient by questions, and are not always given by him spontaneously. Malignant sarcomatous growths must always be carefully sought for, and the state of the spleen and lymphatic glands must be investigated. If there be such an affection as a "myelogenous leuchæmia," its presence can hardly be negatived in any other way than by a microscopical examination of the blood.

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* [In a case which died under my care in 1883, Mr Watson Cheyne found in the tissues, strings of micrococci. In a case of Dr Russell's, of Carlisle, he had previously found bacteria. 'Path. Trans.,' vol. xxxv, p. 408.—Ed.]

DISEASES OF THE SKIN

(*BY DR. PYE-SMITH*)*

INTRODUCTORY CHAPTER

General pathology—Classification—Nomenclature—Historical sketch—The elementary anatomical lesions—The local distribution of cutaneous affections: in depth (bathymetric) and over the surface (regional)—The circumstances: age and occupation of patient, season, history, subjective symptoms and disturbance of other organs.

The fact that many eruptions of the skin are closely attendant upon febrile and other general disorders early attracted notice; and the humoral pathology which pervaded medicine from classical times until almost the present day afforded a ready explanation of their occurrence. Hence cutaneous diseases were long regarded as mere symptoms of some hypothetical "dyscrasia" of the humours; when the Galenical doctrine of the four humours was given up they were thought to be due to disorders of the blood; then, when better knowledge of the chemistry and morphology of the blood began to stand in the way of such easy explanation, they were ascribed to "diatheses," or tendencies, of which the eruption was at once the evidence and the effect. Explanation by assumed causes still took the place of inquiry into anatomical and clinical facts.

Beside an antiquated and false pathology, another obstacle to the advance of knowledge in this department of medicine has been the continuance of the absurd nosologies, the artificial and cumbrous classifications, and the pseudo-scientific nomenclature which formerly encumbered the whole domain of medicine and still linger in this corner of the field.

General pathology.—It is now well known that diseases of the skin differ in their origin and significance no less than diseases of the tongue, the eye, or the bladder. Some of them are symptomatic of specific febrile diseases, of which they form a part, and are therefore important chiefly for the sake of diagnosis. Others are produced by certain articles of food, by poisons, or by drugs. Some are examples of various pathological processes which are familiar in other organs, as cancer, hypertrophy, atrophy, hæmorrhage, pigmentation. But the majority are examples of the widespread morbid process called inflammation, a process which is sometimes the result of definite local irritation, but often independent of such obvious cause, and therefore at present called primary or idiopathic. In this and in other respects they are analogous to the inflammations which affect the bronchial mucous membrane, the stomach, the intestines, and the urinary tract. Closely resembling these structures in its origin and development, its general anatomy, its vascular and nervous relations and its glandular apparatus, there is no doubt that the skin has also a pathological alliance with the mucous membranes.

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* This subject, from the present page to the end of the volume, had not been begun by Dr Fagge, and is therefore added by the Editor.

Diseases of the skin are best studied as examples of general pathological processes, modified only by the peculiarities of the affected tissue.

Classification.—Any arrangement of diseases is valuable so far as it helps the memory to retain useful facts; any arrangement is useless or mischievous if it pretend to be a universal or "natural" or "scientific" system. Diseases are not natural objects; they are physiological states, which we sometimes define by their cause, as plumbism and scabies, sometimes by their histology, as sclerosis of the cord and epithelial cancer of the lip, sometimes by their constancy in transmission, as measles and typhus, and sometimes as a more or less constant collection of symptoms, as chorea and epilepsy.*

Nomenclature.—We must abandon the binomial terminology which once extended to the whole of medicine, as founded upon a misleading analogy between natural organisms like plants and animals and heterogeneous objects like diseases. Next to false pathology and fruitless attempts at classification nothing has been more injurious to the rational study of this group of diseases than a cumbrous, pedantic, and often barbarous nomenclature.

Historical sketch.—The point of view from which diseases of the skin were regarded almost down to the present century was, as we have stated, the hypothesis of the four Galenical humours: the blood which took its origin in the liver, the phlegm secreted by the pituitary gland, the bile by the gall, and the black bile by the spleen. From due mixture of these humours arose the four natural temperaments: sanguine, in which the blood was predominant; phlegmatic or pituitous; bilious or choleric; atrabilious or melancholic; and from their ill-mixture resulted dyscrasiæ, such as scurvy, scrofula, and gout. During the eighteenth century the causes and indications for treatment of an eruption were readily determined by learned physicians to be due to a strumous cachexia, or a scorbutic state of the blood, or vitiated humours from obstruction of the primæ viæ, or lues venerea, but the actual physical conditions of the skin were scarcely noticed. It is the great merit of Willan (and in some degree of his predecessor Plenck of Vienna) that he accurately described the *anatomy* of the morbid skin. His "orders," the *elementary lesions* of later dermatologists, are the alphabet of the subject, and correspond with the "physical signs" of diseases of the lungs introduced by Auenbrugger and Laennec.

From the English or anatomical school of Willan and Bateman sprang the French school of dermatology, which may almost be described as *ætiological* in aim. Biett, its founder, was a pupil of Willan, and introduced his system into France. He had the great merit of perceiving that syphilis does not merely act along with other predisposing causes in producing diseases of the skin, but that it has as its direct consequence definite, constant, and recognisable lesions, the recognition of which is all-important for diagnosis and for cure. He was succeeded by Cazenave (1843), Devergie (1854), and other systematic writers, who continued the work of clinical investigation and accurate description. Unfortunately Biett's success in tracing certain cutaneous affections to syphilis led to the formation of similar groups of "scrofulides" and "maladies dartreuses."

The attempt to define cutaneous diseases by their true *nature and cause* instead of by anatomical lesions had been already made by Alibert, a contemporary of Biett. His eloquence and power of picturesque description had much influence, which was increased by the publication of a magnificent atlas of plates, illustrating his 'Arbre des Dermatoses.' His pathology, however, was erroneous, his descriptions superficial, his nomenclature inaccurate, capricious, and "provincial." The same attempt to explain rather than to investigate, and to supply the nature and causes of disease by hypothesis when proof is absent pervades the voluminous writings of Bazin (1853—70), who carried the hypothesis of "diathesis" to its extreme limit. The same principles are illustrated in the interesting and clinically useful lectures of Professor Hardy (1858—64), so long connected with the great hospital of St Louis.

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* This subject is dealt with by the present writer in the twenty-second volume of the third series of 'Guy's Hospital Reports' (1877), where examples of classification of diseases of the skin are given on the several bases of pathology, ætiology, anatomical distribution, prevalence at certain ages, and reactions to remedies (pp. 174—177).

Meanwhile, another school had arisen at Vienna which was guided by the physiological doctrines of Rokitansky. Its founder was Ferdinand Hebra, whose writings have done more than those of any other man to put the study of dermatology on a sound basis and to extend its limits.*

The *pathological* school of Vienna represented by Hebra, the *diathetic* school of Paris represented by Hardy, and the *anatomical* and *therapeutical* school of England represented by Erasmus Wilson (1847—67) have all changed during the last twenty years. *Histological* investigation by the improved methods of the last twenty years have thrown much light upon the morbid processes of the skin, and the newest school of dermatology, that of America, is making important contributions to the subject.

Before entering upon the description of the several diseases to which the skin is subject it will be well to say a word on the three most important elements in their description and diagnosis.

I. THE ELEMENTARY LESIONS.—The following are the most characteristic and important:

1. *Hyperæmia* or Congestion.—(*a*) Mere overfulness of the vessels from paralysis of the vasomotor nerves with redness and heat, but without the exudation and tissue changes which accompany inflammation. This hyperæmic blush, readily produced as a physiological event in the physiological laboratory, is rarely seen as an uncomplicated morbid condition (*e.g.* Trousseau's 'Tache Cérébrale,' see vol. i, p. 588).

(*b*) *Active inflammatory hyperæmia,* varying in colour from brilliant scarlet to rose pink and combined with heat, tingling, or other sensations. Such an early stage of inflammation is often called "erythema." The local swelling and the subjective symptoms distinguish it from the non-inflammatory hyperæmia just described.

(*c*) *Passive,* venous or congestive *hyperæmia,* dependent upon retarded circulation and distended venules. The colour is purple, bluish, or livid, surface cold, and there are no painful sensations. This passive congestion, frequently seen as the result of thrombosis and also in chronic affections of the heart and lungs, is often associated with the more chronic forms of inflammation in which œdema is present and connective-tissue overgrowth is apt to result. The best example of this condition is in the congestive erythema of "chilblain."

2. *Pimple or Papule.*—A solid small elevation of the skin. Under this name more than one pathological lesion is included.

(*a*) The inflammatory papule, more or less pointed, bright red, small, and very early seen with a lens to contain a minute drop of exudation, either abortive or ending in a vesicle or pustule. Another variety of solid non-inflammatory papules are those which consist of true hypertrophy of the normal papillæ of the cutis. These form the minute multiple warts which occasionally occur in immense numbers; more common are local warts and condylomata.

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* His 'Acute Exantheme und Hautkrankheiten' was the third volume of the series of textbooks of 'Pathology and Therapeutics' superintended by Virchow, and was published in parts between 1860 and 1874. Much of the latter part is written by Hebra's son-in-law Moritz Kohn, afterwards Kaposi. The translation into English for the New Sydenham Society (1866—75), begun by Dr Fagge and myself, was completed by Mr Waren Tay.

(*b*) Chronic large papules, never showing liquid exudation, but apt to become covered with minute scales. Sometimes, as in psoriasis, papules increase so as to form a raised patch, and then become covered with scales or coalesce with the same result as in lichen planus. More often the papules remain discrete and without scales as in prurigo.

(*c*) Solid elevations of the skin, which may be called false papules; such as the heaped-up scales at the orifice of a hair-sac which form the so-called "lichen or pityriasis pilaris," and a sebaceous gland occluded by its own secretion which is called a "comedo."

3. *Vesicle.*—A visible cavity in the skin filled with transparent liquid. In almost all cases the vesicle is inflammatory, and the liquid is exuded plasma consisting of water, salts, albumen, and a few leucocytes with only a trace of fibrinogen. Where the epidermis is thin the vesicles rupture almost as soon as they form, but where it is thick, as in the palm and sole, they grow and coalesce into large bladders. Broad and flat vesicles, as those of zona, are usually distinguished from the smaller and more closely packed vesicles of eczema. The vesicles of smallpox are remarkable not only for their size and depth, but for the exudation being so effused into the meshes of the papillæ and Malpighian layer that the cavity is "pocketed" and shows a central depression or *umbilicus.*

Non-inflammatory vesicles consist of retained excretion either of sweat-glands (sudamina) or of mucous glands. The latter are practically the only vesicles seen on the mucous membranes, for under the moisture and friction of the mouth, though inflammatory vesicles form, they are scarcely ever seen before they burst.

4. *Pustule.*—A cutaneous abscess, that is, a cavity in the skin containing inflammatory exudation, water, salts, albumen, and abundance of dead leucocytes in a state of fatty degeneration, with usually only traces of fibrinogen. The distinction between a vesicle and a pustule is therefore usually one of time only and rests upon the abundance of the corpuscular element in the exudation; but while most vesicles become pustules the exudation remains serous in many cases of eczema. Again, in contagious impetigo, in furunculi, and in some other cases, the first visible exudation is opaque, yellow, and purulent.

5. *Bulla* or *Bleb* is the name given to a very large vesicle. It is always inflammatory and of essentially the same pathology as a vesicle or pustule. It contains at first transparent serum and this usually becomes more or less completely purulent. There are also almost always shreds of fibrin to be seen.

6. *Scab* or *Crust.*—A dried-up concretion of the contents of a vesicle, pustule, or bleb. Its form depends upon the inflammatory process ceasing, otherwise fresh exudation succeeds and no dried-up mass is allowed to form. The size of a scab will always depend upon that of the pustule or bleb which formed it, its thickness upon the amount of fibrin and leucocytes in the exudation. Its colour is often characteristic; light brown or yellow when the exudation is serous, deeper yellow (which the older anatomists compared to honey in the term *Porrigo favosa*), greenish yellow in some cases where the pus is thick, red or almost black where the exudation contains red blood-discs.

7. *Scale* (*Squama*).—A dry flake of epidermic cells. When scales form in moderate amount and of small size as the result of inflammation which has passed, they are described as *furfuraceous*; when large, adherent, imbricated, and glistening silver-white from the refractive power of air enclosed in the spaces, scales have the characteristic appearance seen in psoriasis.

Large, thin, and very abundant scales, which have been compared with dry hop leaves, which are sometimes termed squamous, are almost characteristic of pityriasis rubra. Beside the true epidermic scales desquamation often consists of dried-up sebum or of dried exudation mixed with epidermis. The microscope distinguishes the amorphous fatty material of the former and the leucocytes of the latter from the flat horny cells of true scales.

8. *Wheal* (*Pomphos*).—A flat solid elevation of the skin much larger than a papule, and of ephemeral duration. Such wheals may be either traumatic or idiopathic, they are the characteristic effects of the poison of the stinging-nettle and of the form of erythema, hence called urticaria. They probably consist of acute œdema of the skin producing local anæmia by pressure.

9. *Scratch-mark.*—An injury to the skin of linear form and curved outline, usually marked by dried-up blood, and having a definite relation to the range of the patient's hands. They are of diagnostic value as proofs of pruritus.

10. *Raw.*—A surface which has lost its horny layer of epidermis so that the moist and living Malpighian layer is exposed, from which more or less exudation oozes. Such a raw weeping surface is characteristically seen after pricking the blister formed by cantharides. It also results from the rapid rupture of a number of vesicles as in the kind of dermatitis called eczema madidans.

11. *Chap* (*Rima*).—A crack or fissure which goes through the epidermis to its Malpighian layer or to the vascular papillæ beneath. These rimæ or rhagades sometimes extend very deeply, are apt to bleed, and are always extremely painful.

12. *Sore* (*Ulcus*).—The result of destruction by inflammation which has reached below the Malpighian layer and has destroyed the papillæ; characterised by the absence of any trace of epidermis, by the granulations which cover its floor, and the pus in which they are bathed.

13. *Scar* (*Cicatrix*).—The result of the healing process after an injury or disease which has been deep enough to destroy the papillæ of the part. Accordingly the presence of a cicatrix, however superficial and slight, shows that the preceding process affected the deep layer of the cutis.

14. *Nodule.*—A solid elevation of the skin larger than a papule, and seated in its deep layer. The nodule was formerly called a tubercle, but the word "tubercle" should never be applied except with its present pathological meaning. A *node* is a large nodule, and there is no reason for restricting the term to syphilitic nodes or *gummata*.

15. *Stain* (*Macula*).—A patch of increased pigmentation of the skin, either the result of long-continued preceding hyperæmia or occurring independently as a primary increase of pigment.

II. Distribution.—After determining the morbid anatomy of a disease of the skin the next step is to notice its distribution.

(A). *In depth.*—In its pathology the skin does not follow the anatomical and embryological division into epidermis and cutis vera. It may be physiologically divided into three layers :

(1). The *horny layer of epidermis* or *cuticle*, dead scales, the only affections of which are increased growth, atrophy, dryness, desquamation, and other results which really depend upon perverted growth in the living layer of cells which lies immediately beneath it.

(2). The *living Malpighian layer* of the epidermis, together with the *papillary layer* of the cutis. These two tissues are constantly and inseparably united in their pathology. Their inflammation constitutes the enormous group of diseases which come under the head of superficial dermatitis. Affections confined to this part never leave scars.

(3). The *deep layer of cutis* with the *subcutaneous connective tissue.* Inflammation or new growths beginning below the papillæ are prone to spread to the subcutaneous tissue and not to stop until they reach subjacent muscle or bone or deep fascia. The deep affections of the skin which lie in this region are less numerous, but more severe, than those of the superficial layer, and are always marked by cicatrices.

(4). Lastly come the cutaneous affections which particularly affect the *sweat-glands,* the *sebaceous glands,* the *hairs* and hair-sacs, or the *nails.*

(B). In their distribution over the *surface* of the body the diseases of the skin differ greatly. The earliest attempts at classification were between affections of the scalp and of the trunk. In Willan and Bateman's system, this character did not receive due consideration ; but it has, I think, met with still less at the hands of French and German dermatologists. Even in the best descriptions of Hebra and his successors it is sometimes impossible to learn what part of the body is affected by a particular disorder. But the fact is that very few diseases of the skin are indiscriminate in their extent, while many are at least as definitely and exclusively fixed to certain localities as the lesions of enteric fever in the intestine, those of tubercle in the lung, or those of tabes in the spinal cord. The skin is not uniform in its structure, the relative thickness of its layers, its vascularity, its nervous supply or the distribution of its glands. Its different parts are variously protected both by natural and artificial coverings, they are variously exposed to injuries, to irritants, and to moisture. It is therefore not surprising that their diseases differ so greatly. Psoriasis of a flexor surface and scabies of the face should be regarded, like carcinoma of connective tissue or phthisis of the base of the lung, as altogether exceptional. It must be remembered that in childhood the several regions of the skin are not yet differentiated, and hence the local distribution of its diseases is less strictly adhered to than in adults. We find precisely the same rule in the distribution of pneumonia, of malignant disease and of tubercle, in children. The several regions will be fully described in the following chapter on eczema and dermatitis generally.

III. The third group of characters includes all the *circumstances* of a case of cutaneous disease. They help in diagnosis, they throw light upon pathology and causation, they frequently supply hints for treatment. We have to consider :

1. *The age* and *sex* of the patient. Some affections, like prurigo pedicularis, are scarcely seen except in the aged skin ; others, like impetigo, are extremely common before puberty and extremely rare afterwards ; others, like true acne, are with rare exceptions confined to the period of adolescence.
2. *General health* and particularly the state of the stomach and bowels, the urine, and the temperature.
3. *Occupation* and external circumstances which affect the surface of the patient's body, including intercourse with other persons affected with a similar disease.
4. *Climate,* season, temperature, moisture or dryness of the air, sun, frost, and wind.
5. *History* of the malady, its duration, the manner of its onset, and particularly, when obtainable, a knowledge of the primary lesion. The fact of recurrence is also of great importance.
6. The *subjective symptoms ;* pain or discomfort, itching, burning, smarting tenderness, or neuralgic pains, alleviation by exposure to the air, or by covering, by heat or cold, by the application of water or oil, by pressure or by friction.

ECZEMA

(*MOIST TETTER*)

AND COMMON SUPERFICIAL DERMATITIS

Definition—Willan's—Hebra's—Its distinction from other forms of dermatitis—Histology—Anatomical lesions—Course—Distribution and local varieties — General symptoms — Ætiology — Diagnosis — Prognosis — —General treatment—Local applications—Diet and regimen, baths, &c.—Internal remedies—Special treatment of local varieties.

IMPETIGO.—*Its relation to eczema—to pediculi—to contagion—Its treatment.*

By far the most important of diseases of the skin from its frequency, and perhaps the most important of all diseases which do not shorten life, from its obstinacy and the misery it occasions, is the affection now universally known as eczema (ἔκζεμα), the "outbreak" or "eruption," as the Greek physicians called it, the "moist tetter" of our forefathers. In its commonest form it is familiar to the profession and the public, and cannot escape instant recognition, but under many circumstances it is difficult to diagnose, and opinions have differed widely as to its pathology, its definition, and the extent to which dermatoses bearing other names are allied to or identical with it.

Definition.—Willan classed eczema among *vesicular* diseases, and this is a proof of his acumen and judgment; for, although the vesicles of eczema are so small and numerous, so short-lived and speedily supplanted by pustules or weeping surfaces or scales that one may see hundreds of cases before the vesicular stage can be demonstrated, yet there is no doubt that vesicles are characteristic and, if not a constant, the most nearly constant anatomical lesion of eczema.

The most important step in the pathology of this disease was Hebra's statement that eczema can be produced at will, for it is in fact identical with the *common superficial dermatitis* which is the result of ordinary irritants. As a result of this important statement Hebra not only described under eczema erythema, intertrigo, the pustular form of dermatitis known as impetigo, most cases of papular dermatitis previously classed under various species of lichen and strophulus, but he boldly included scabies itself as also a common inflammation of the skin and therefore a true eczema. All succeeding dermatologists have more or less followed Hebra in extending the bounds of eczema far beyond the definitions of Willan and Bateman.

But invaluable as the new doctrine of Hebra was it has become clear that for clinical purposes we must seek again to narrow the definition of the word eczema. Inflammation, the reaction of the living tissues to injury, is the key-note of pathology. If to the doctrine of inflammation we add that of degeneration and new growths, of parasites and of contagia, almost the whole range of modern pathology is covered. It is quite true that the vast majority of diseases of the skin, like those of the rest of the body, are inflammatory, but for prognosis and cure we need much more than this elementary fact. Hebra himself had too much sagacity and practical sense to be led far astray by his own reform.

(1). Syphilitic diseases are most of them undoubtedly inflammations of the skin, but however closely they might approach in symptoms and appearances to some forms of eczema he separated them very widely. Scarlatina is a dermatitis not unlike some stages of eczema. Variola and varicella often approach impetigo still more closely in appearance, but neither Hebra nor any of his disciples have classed the exanthemas of Willan with eczema. These diseases are all separated by our knowledge of their ætiology, by their combination with definite symptoms in other organs than the skin, by their course, and by the practical measures for which they call. (2). Scabies, again, is distinguished from all other dermatites, not by the pathological process, but by the peculiarity of the irritating agent, by the consequent characteristic distribution, and by the special mode of treatment. (3). Lastly, we must separate from true eczema diseases like psoriasis, which, though undoubtedly inflammatory, are special in their characters, in their anatomy, in their chemical products, in their results, and (above all) which cannot be producėd or even simulated by an external irritation. In other words, they are not "common superficial dermatites" such as corresponds with the natural reaction of the healthy skin against a common, mechanical, thermal, or chemical irritant.

But now comes a more fundamental definition of the term which I believe to be absolutely necessary for those practical objects which are after all the end and justification of all refinements of nomenclature. If we call eczema common superficial dermatitis and assert with Hebra that we can produce eczema at will by rubbing in an irritant ointment or by exposure to the sun, we run the danger of forgetting what is after all a most important character of the disease which we all agree to call eczema, whatever else may be included under the name. Undoubtedly "wet tetter" is in the majority of cases *not* the direct and immediate result of local irritation. I therefore prefer to say that a scorching sun or a mustard plaister will often produce a common superficial weeping dermatitis which is histologically and chemically absolutely identical with eczema, which may, if we please, be called artificial or traumatic eczema, but which yet differs from the true disease by the very fact that it is the physiological reaction of the healthy skin to a definite known irritant; that it further differs in its course, in its distribution, in its whole natural history from idiopathic, typical, true eczema, and demands as a consequence a different prognosis and different therapeutics. The distinction, however subtle in theory or difficult to draw in practice, is of direct and paramount importance.

For instance, the surgeon of a gaol is shown an eruption on a prisoner's arm, which, by every anatomical character is a "common superficial dermatitis"—is what might have been made by Hebra's eczema-producing liniment. He diagnoses eczema, prophesies the course it will take, its obstinacy and its probable recurrence, and prescribes what he calls appropriate treatment. But whether verbally correct or no he has made as great a practical blunder as is possible. The common superficial dermatitis was traumatic, the eczema was not like that produced by an irritant, but was actually and designedly so produced; the subject of it is not a patient with a disease, but a skilful impostor who has inflicted injury upon his skin; the course of the eruption will not be guided by the natural history of eczema but by the will of the patient, it will not recur except by his wish, and will not be cured by "appropriate treatment." Since, therefore, the name which follows a diagnosis should connote as much knowledge as possible in brief, I think it much better not to name factitious dermatitis "eczema."

In the same way I would exclude all common superficial dermatitis which is the direct and immediate result of local irritation, for what is remarkable is not that the skin should inflame when irritated, but that the skin of many people is liable to undergo the exact pathological changes produced by irritation *without* any demonstrable irritant.

Eczema, therefore, I would define as "idiopathic, common, superficial dermatitis." I fully admit, however, the difficulty or impossibility of drawing a line in every case. We can only classify diseases as they more or less naturally are connected with certain typical forms. At one end of the scale we have purely traumatic dermatitis produced by a demonstrable external irritant, limited to its immediate effects and disappearing not to return when the cause is once removed; at the other end we have dermatitis appearing on parts of the skin which are not exposed to any known irritation, following a distribution which is independent of irritants, recurring without external cause after it has once disappeared, and only curable by measures other than those addressed to the local irritation. But in every case of dermatitis, however idiopathic, there is no doubt an *irritans* if we could only recognise it, and in every case, however traumatic, there is an *irritabile* in the patient's tissues. Inflammation can never be truly "idiopathic," that is uncaused; for like every other event it depends upon antecedents. No heat of the sun, no activity of cantharides or of croton oil can produce a pustule or a bleb upon the skin of a corpse. All eczema is common superficial dermatitis, but every common superficial dermatitis has not the characters in its origin, its distribution, and its course, in fact in its whole natural history, which entitle it to the name of eczema. In order practically to identify eczema we must therefore look for the clinical characters to be presently described.

Eczema is dermatitis at the stage of *exudation:* it is well called "moist tetter." Cases of dry eczema no doubt occur, but they are either abortive or residual. When we use the term eczema, we imply that the eruption is moist, or will be moist, or has been moist; or that at least it occurs in a person who has previously, or will hereafter, be subject to another outbreak of the same thing where exudation will be apparent.

Slight degrees of inflammation, when the result of irritants, fall under the minor degrees of superficial traumatic dermatitis. Slight degrees of idiopathic inflammation which do not reach the stage of exudation—hyperæmia, roseola, erythema of the skin—when not shown to be abortive eczema by their locality and course, belong to very different pathological groups. They may be, first, symptomatic rashes like those of measles and scarlatina which are true dermatitis with all the characters of inflammation, and followed by desquamation. Under the same head of exanthemas I would include, secondly, the roseolar, erythematous or papular rashes of enterica, cholera, and syphilis. Again, there are superficial forms of dermatitis which never assume the characteristic exanthematous aspect, which differ entirely in locality, in the persons they are most prone to affect, in their local and general symptoms, and in their constantly subacute character. These superficial dermatites, of which erythema-nodosum is perhaps the best type, are clinically and pathologically to be separated from eczema, and will be treated of in a separate chapter.

Histology.—The pathology of eczema is that of inflammation generally. Its signs are the four Galenical characters of pain, heat, redness, and swelling, to which we now add a fifth, pyrexia or febrile reaction. Of its cause we know no more than of inflammation in other parts. Traumatic inflammation follows injury or death; idiopathic inflammation we assume must have some corresponding lesion, but of its nature we are ignorant. The order of events is vaso-motor paralysis, dilatation of the small arteries and capillaries, stagnation of the blood-stream, diapedesis of leucocytes through the stomata of the capillaries, and exudation of the plasma or liquor sanguinis.

If a section of eczematous skin be made the cuticle is found unaffected, the Malpighian layer swollen, the papillæ œdematous with dilated blood-vessels, and multitudes of leucocytes clustered round them; the deep layer of the cutis and subcutaneous tissues are unaffected. Looking at the living skin we see, so soon as a sense of slight irritation with some pain of a tingling or smarting character had drawn the patient's attention to the spot, that there is already an inflammatory blush. This usually has from the beginning a brighter, more arterial hue than the rose-coloured tint of true erythema. A more important distinction is that the erythematous blush is diffused and fades off at the edge. It is scarcely ever disposed in blotches, circumscribed or mottled patches or figures of definite outline. The swelling from œdema is very slight. On close inspection, particularly if a lens be used, one can see that the apparently uniform redness is produced by a number of isolated deeper-coloured points. In this and in other respects the early stage of eczema resembles scarlatina as true erythema resembles measles.

Before long, but never without a precedent stage of hyperæmia, there appear minute vesicles. Frequently, however, they are preceded by little red elevations, which for some time show no bright transparent spot of fluid, and such inflammatory papules may appear early and continue for a long time before becoming vesicular. Such papular forms of eczema must be regarded as abortive, and very seldom will a careful scrutiny fail to discover the evidence of liquid exudation at one period or another of the case. Soon after the vesicles have formed the remarkably thin roof of the cuticle ruptures and they run together, forming a raw weeping surface, eczema madidans, or they may previously have sunk somewhat deeper and acquired more or less purulent contents before their thicker roof bursts. Such pustular forms of eczema usually produce, not weeping surfaces, but more or less extensive scabs, though intermediate stages are very frequent. In the most typical form of eczema the weeping stage continues until a great abundance of clear watery exudation is poured out. It consists chiefly of serum, to which the salines give its irritating property and the albumen its characteristic effect in stiffening linen. On the raw weeping surface it is easy to distinguish more injected points which mark the seat of ruptured vesicles. This *état ponctué*, as the French writers call it, is very characteristic and may be sometimes seen before and even after the moist stage. The involution of eczema is accomplished by the exudation diminishing, and at last drying up, the weeping ceases, or scabs take the place of pustules. Finally, the cuticle again covers the abraded surface and a branny desquamation, formerly described as psoriasis or pityriasis diffusa and also as eczema squamosum, covers the lately inflamed part. The itching still continues and is sometimes troublesome up to the very last.

In chronic eczema the skin becomes extremely thickened, which is readily appreciated on pinching up a fold. It is constantly covered with branny desquamation, acquires a deep reddish extent of a brilliant scarlet colour, and in certain parts is marked by deep fissures or rhagades, which often penetrate to the true skin and give rise to bleeding and excessive pain. This eczema rimosum is most frequent in the palms or the soles and in its hæmorrhagic form on the nipples and the lips.

Course.—The course of eczema is very rarely acute in origin, development, and recovery; even in what appear to be the most acute cases it will be found that the patient has been subject to previous attacks, and that in the intervals small patches of the disease linger behind the ears or on the face or hands or some other isolated part, and when the acuteness of the attack has passed off, it is rare for the whole of the skin to return to its normal condition. Fresh smaller outbreaks occur again and again, so that with scarcely an exception, however acutely the first attack of eczema may sometimes begin, its course is a chronic one.

Another peculiarity also very characteristic of eczema is its strong tendency to *recurrence.* It is extremely rare for a person to suffer from a single attack in the course of his life. Again and again when the disease appears in a quiescent condition a fresh acute outbreak will occur or one attack will scarcely have passed off when another supervenes. Happily the majority of cases are not lifelong in duration, but they usually extend over several years, and it is not unusual for recurrence to take place even after long intervals of complete freedom.

Distribution.—One of the distinctions between eczema or idiopathic dermatitis and that which is traumatic in origin is that while the latter corresponds more or less exactly to the irritant, typical eczema has its own peculiar laws of local distribution. Speaking generally it is a disease of the thinner parts of the skin, it is a disease of the flexures and joints, it is a disease of the head and limbs rather than of the trunk, and lastly it is a symmetrical disease.

I must stop for a moment to explain the meaning of the word *symmetry* in pathology. It is nothing to the point to call universal eruptions like that of scarlatina symmetrical; they are so only because the human body is itself bilaterally symmetrical. Nor is it enough that the same diseases should be found in both right and left member as in acute rheumatism. Symmetrical distribution means that exactly the corresponding parts on the right and left side are simultaneously affected, both ears, both elbows, the back of each hand, the under surface of each wrist and the popliteal space on each thigh, the sole of each foot. This is bilateral symmetry, but we also see examples of serial symmetry in pathology where the same condition is seen on the elbow and the knee, the wrist and the ankle, the palm, and the sole. Eczema is an extremely symmetrical disease, as thus defined, more so than any other affection of the skin excepting psoriasis.

The most characteristic locality for eczema is behind each *auricle,* not only because it is so frequently seen here when it affects other parts, but also because this spot is but little liable to other diseases. The *face* is more frequently affected with the pustular form of eczema (which we shall presently describe as impetigo) than any other part of the body, and this particularly applies to the lips, nostrils, and cheeks. In adults the face is less frequently the seat of ordinary eczema, coming next in frequency to the *limbs.* The same remarks apply still more strongly to the *scalp,* where impetigo is the commonest affection in childhood, but where ordinary eczema is comparatively rare except on the bald scalp of infancy or age. Eczema does not frequently affect the skin which is covered by the *beard,* and when it does is not usually remarkable for obstinacy, but sometimes the inflammation can be unmistakably seen to penetrate to the hair-sacs and there become a deep instead of a superficial dermatitis. Its clinical features and treatment are then so different that it is properly known as a spreading disease, sycosis. It may in like manner spread to the sacs of the *eyelashes* and become localised as what used to be called tinea tarsi.

The *neck* is very frequently the seat of eczema, especially the front and sides. On the *trunk* the shoulders, back, and loins are but rarely the seat of the disease, and the same applies to the gluteal region which is so frequently the seat of isolated pustules not only in scabies, but in the impetigo and ecthyma of children. The flanks, though covered with soft and delicate skin, are not often affected with eczema which when present has usually spread from the axillæ or from the neck; and the same applies to the chest. In women, however, eczema of the *breast* is common either as eczema intertrigo beginning in the fold under the mammæ or as eczema of the nipple. The *abdomen* is more often the seat of eczema than other parts of the trunk, especially of that variety which begins at the *navel.* The *genital organs* by the thinness of the skin and mutual contact are readily disposed to dermatitis, but are certainly less often affected with ordinary eczema than either the face or the limbs. Either the inflammation begins as intertrigo of the scrotum and thigh, especially common in infants or it is an acute weeping eczema which extends to the abdomen, thighs, and other parts as well, or it is a chronic and extremely pruriginous eczema of the vulva or scrotum. The last form even more frequently affects the neighbourhood of the *anus,* particularly in elderly persons: and the cleft of the nates is extremely liable to eczema intertrigo, both in infants and in fat persons, under the irritation of long walking and free perspiration. In many cases eczema of the anus, perinæum, genital organs, and thighs forms a well-marked local variety, which must be carefully distinguished from the so-called "eczema marginatum" of the same regions, to be afterwards described as a form of tinea.

On the *arms* eczema is scarcely ever seen over the deltoid, and though seen in the axilla, and particularly its anterior fold, is much less so than at the elbow. The bend of the elbow is probably, next to the face and ears, the most frequent seat of ordinary eczema, and if we were to exclude cases occurring under puberty even that exception need not be made. The skin covering the biceps cubiti and the flexors of the front of the forearm usually participates in eczema of the elbow, and this local form of eczema is one of the most constantly and accurately symmetrical. The disease scarcely ever affects the olecranon, but there is a form of eczema which, though relatively uncommon, is seen quite often enough to deserve special mention. It is an ordinary weeping eczema occurring in adults and affecting the outside of both forearms from an inch or more below the point of the elbow down to the wrist. It is extremely symmetrical and often affects the skin of the upper arm which covers the triceps though without spreading to either shoulder or elbow. The *wrist* and back of the hand are comparatively seldom the seat of eczema, though the last-named will sometimes spread as far as the knuckles, and this region is not unfrequently the seat of the dry chronic circumscribed dermatitis which I shall describe as single patch eczema. The *fingers,* especially their clefts, are often the seat of eczema, but in most cases this can be traced to a traumatic origin. The *palm* of the hand might seem little adapted to eczematous inflammation from its thickness, but it is very frequently the seat of a characteristic chronic dermatitis painful, disabling, symmetrical, and obstinate, which, from the absence of vesicles and the presence of deep fissures dependent upon the thickness of the epidermis has received the name of eczema rimosum. This is, however, either of local traumatic origin or at least is usually unassociated with eczema of other parts, and is curable by local applications alone. Eczema of the matrix of the *nails* is almost always part of eczema manuum. It proves a long period of dermatitis, and its presence is therefore a point of diagnosis in distinguishing eczema from scabies. The consequent malformation of the nail is generally marked by longitudinal grooves, and by less thickening than in the far more rare psoariasis unguium.

In the *lower extremities* eczema of the groin and inner part of the thigh is very common in adults, and is either associated with eczema of the arms or with eczema of the abdomen and genitals. The outside of the thigh is not often the seat of ordinary eczema and the patella, like the olecranon, is practically exempt, but the popliteal space is almost as favourite a seat of the disease as the bend of the elbow, and the inflammation spreads thence more or less extensively over the thighs and legs. Below the knee, however, eczema is on the whole less frequent than below the elbow, and most often appears in one of two forms. (1) Eczema of the calf and peroneal region of an ordinary weeping and irritable type frequently accompanies the corresponding affection just described of the outside of the forearms, and, like it, is almost always symmetrical. (2) Varicose eczema, a local dermatitis often unconnected with eczema of other parts, is obviously the result of ordinary irritation acting upon a skin congested by varicose veins. It affects the inner side of the leg from the internal malleolus upward, that is to say from the point where, as Hilton showed, the least considerable anastomosis takes place, between the internal saphenous and the posterior tibial veins. This form of dermatitis is known by its purplish tint and frequent association with ulcers as well as by its locality. The foot is less often affected with eczema than the hand, but follows its serial homologue very closely. When not of traumatic origin and not due to scabies or to intertrigo, that is to say, when dermatitis of the *foot* is true eczema, it either affects the dorsum in association with eczema of the outside of the leg or the soles as a chronic eczema rimosum much resembling that of the hand, or it is an eczema intertrigo digitorum sometimes leading to deep clefts between the toes. This last form is as common, if not more so, than eczema of the fingers. It is rarely associated with the disease in other localities, and must be treated entirely by topical measures.

Symptoms and natural history.—Locally, eczema provokes extreme itching, more so perhaps than any other cutaneous disease except prurigo and scabies. Indeed, although unlike these two there are many cases of eczema which are almost free from irritation, yet in others the pruritus seems to be at least as intense, constant, and obstinate a symptom as in the worst of any other disease. Itching is usually less in the weeping and acute than in the dry, papular or chronic and scaly conditions, and it is very rarely marked in the pustular form of eczema. It is most intense in ordinary eczema of children, and in that of old persons, and of all local varieties is most constant and most severe in eczema ani et vulvæ.

Smarting, tingling, and some amount of local tenderness are common symptoms of the more acute and ordinary forms of eczema, and are associated with a peculiar sense of burning and tension. There is rarely more than slight febrile movement at the onset of the disease and particularly in large surfaces invaded at once, but even when the thermometer shows no appreciable elevation of temperature there is thirst, loss of appetite, and a slightly furred tongue. The mucous membranes are unaffected ; there is no foundation for such names as eczematous gastritis, enteritis or bronchitis. The pathology of the digestive, pulmonary, and urinary mucous tracts is quite different from that of the skin. We have no right to assume an eczema of membranes which we cannot see when we cannot demonstrate eczema of those which we can. I have never seen eczema of the lips spread to the mouth or tongue, eczema of the anus to the rectum, eczema of the eyelids to the conjunctiva, or eczema of the penis to the urethra and bladder.

Except for anorexia produced by the slight pyrexia of the onset of eczema, the appetite and digestion are as a rule unaffected in this disease. Dyspepsia is so common that many persons suffering from eczema are also dyspeptic, but there is no reason to regard the latter condition as the result of the former, nor are the bowels either constipated or relaxed. The condition of the urine one would expect beforehand to be affected where the large surfaces of the skin are inflamed, but I am not aware of any satisfactory evidence of any constant change. The fact that in the large proportion of cases of eczema the patients are not confined to bed, or even to the house, makes it the more difficult to obtain observations of the amount, or average condition of the urine in this disease. Any deviation from health which I have observed has been explicable from causes unconnected with the eczema, so that, as far as I know at present, the urine must be stated to be unaffected in this disease.

Ætiology.—I have already stated what appears to me to be the true relation between traumatic dermatitis and idiopathic eczema. When the inflammation directly follows an irritant, is not prolonged after its cessation, does not spread to other than the irritated region, does not recur without fresh irritation, and does not follow the local distribution of eczema, then it is best called common superficial dermatitis of traumatic origin. But some skins, whether by natural stability or by habit, are insensible to sun and friction and sweat, and the other irritants which in ordinary persons produce inflammation. In others a hot day or bathing in sea-water, or an east wind, or a long walk will produce eczema solare or eczema intertrigo or some other form of local traumatic dermatitis—which when once established becomes chronic, persists long after its original cause has ceased to act, and localises itself in the bends of the joints, and in the symmetrical positions which have been above described as characteristic of eczema. Admitting, therefore, irritation of the various kinds alluded to as exciting causes of eczema, we must always admit a certain proneness of the skin to inflammation, and in more than half our cases this predisposition causes the disease without our being able to fix upon any fairly probable exciting cause.

It has been widely supposed that we are to seek the predisposing, and in most cases the efficient cause of eczema in a diathesis or disposition of the whole body, which can be recognised independently of the presence of the eczema, and which produces other recognisable diseases. This diathesis has been called dartrous, arthritic, and gouty, while the ever-ready shelter of a strumous disposition has been invoked when the other failed. I must refer the reader to the works of Hardy and Bazin, of Gigot-Suard, and other French writers, for their exposition of the dartreuse doctrine; while all that can be said for the more particularly gouty relations of eczema will be found in the writings of Erasmus Wilson, Hutchinson, and the late Dr Tilbury Fox. For myself I can only express unbelief in the whole doctrine of temperaments and diatheses, which appears to me to be the residuum of the exploded physiology of Galen, and seriously to impede the advance of medicine. I must here confine myself to saying that although persons with gout are, I admit, often subject to very irritable and obstinate eczema, in the vast majority of cases of eczema there is not the faintest reason for the belief that gout, that is, the deposit of uric acid in the joints, has been present in the patient or his immediate relatives; that there is absolutely no pathological connection between gout and true rheumatism, arthritis deformans, or gonorrhœal arthritis, and that none of these forms of multiple arthritis have the slightest connection with eczema; that eczema rarely co-exists with psoriasis, pemphigus, or other supposed manifestations of the dartrous diathesis; that no one can give an intelligible account of the characters by which this predisposition can be recognised; that there is no evidence that eczema has more than accidental connection with diseases of other parts of the body, or that it is anything but a common superficial dermatitis; and lastly that the diathetic hypothesis is practically misleading in prognosis and treatment, no less than scientifically unsound.*

The efficient cause of eczema, apart from local irritation, is then, I believe, as much and as little unknown as the efficient cause of bronchitis or of cystitis. All we can say is that in some persons the skin is naturally sensitive, or delicate, or irritable. Such persons are, in other respects, like their neighbours, and the predisposition of their skin to inflammation can only be prophesied after the event.

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* I may be allowed to refer to papers on this subject in the 'British and Foreign Medical Review' for January, 1874, and in the 'Guy's Hospital Reports' for 1880.

General eczema is sometimes set up by an accidental and very local irritation, and this is probably the explanation of the undoubted though rare occurrence of vaccination leading to eczema: much more frequently their relation is purely accidental. It appears to me waste of time to discuss the vague speculations, at once unscientific and impractical, which ascribe eczema to such common disorders as dyspepsia, or to such *idola theatri* as constitutional predisposition, assimilative debility, nervous debility, perverted innervation, renal inadequacy, strumous cachexia, scurvy in the blood, acidity of the *primæ viæ*, or diathèse dartreuse.

There is no doubt that eczema is in some cases very decidedly *hereditary*, but it is certainly much less so than psoriasis, and in the great majority of cases there is no reason to admit hereditary predisposition. The exudation of eczema is not contagious so long as it is transparent. When purulent it probably shares in the infective characters more or less common to all pus, and occasionally the pustular eczema which we shall presently describe as impetigo capitis is most markedly contagious.

Eczema affects both *sexes* indifferently. It is common at all *ages*, but differs in its most frequent characters. In the infant it is of the ordinary papular and vesicular kind. The same form is seen in older children, but much more frequent in them is impetigo, or pustular eczema, which is comparatively rare before the first dentition and after puberty. In adults the commonest form is ordinary weeping eczema of the limbs and face. In old age the dry, very chronic, and extremely pruriginous forms are the most characteristic.

Eczema appears to be universal over the globe. It is certainly not more frequent where gout is prevalent, as in London, than where it is almost unknown, as in Vienna and New York.

The traumatic forms of eczema naturally occur in those *occupations* where the hands are exposed to constant irritation. Hence, we have the eczema of the hands which has long been recognised as frequent in washerwomen, grocers, and other hand workers.

It is a popular opinion that skin diseases generally, and particularly eczema, as the most common of them, is most prone to occur at certain *seasons* —the spring and fall. Like all popular beliefs, this,I believe, was not founded upon experience, but chiefly upon theory, and partly, perhaps upon analogy. The period of change in the seasons seems "naturally" to be the most likely period for change in the human economy, and changes are proverbially dangerous. It is possible also that the insalubrity of southern Europe in the autumn, from the prevalence of malaria, led to a belief in the same result in northern countries, while even in England malaria was far from uncommon until quite recent times. However this may be, one does, I think, see eczema of the ordinary irritative and inflammatory kind more often in the spring than at other times, and this may be fairly attributed to the dry east winds which then prevail.

Diagnosis.—Keeping to the definition of eczema as above stated, the only difficulty is on the one hand to distinguish between idiopathic and traumatic dermatitis, or rather to detect the decided and efficient prevalence of a traumatic cause, and, secondly, to draw the line between eczema and certain other forms of superficial dermatitis, the distinctive characters of which, and the justification for their separation, will be considered under each head. The distinction between eczema and intertrigo, eczema and impetigo, eczema and some forms of lichen, and even eczema and scabies, depends ultimately, as all distinctions in medicine should, upon practical utility. What it appears to me we must recognise, is that all superficial inflammations of the skin may be grouped around certain types, and that the most common and important of these, which we call Eczema, is characterised by being *common*, that is to say, the same as is produced by ordinary mechanical or chemical irritants, *idiopathic*, that is to say, not directly co-extensive with irritation, *moist* from visible inflammatory exudence, *symmetrical*, *selecting* certain favourite parts of the skin, and *prone to recur* after disappearance.

It may, however, be well briefly to point out the following characters. From *scarlatina* and other rashes, eczema differs in being never truly universal in its moisture and in being unaccompanied by marked febrile symptoms. From *erysipelas* it is distinguished by the colour, the minute vesicles, the locality and the absence of œdema of the subcutaneous fascia, and, again, by the absence of fever. *Erythema* exudativum is more rosy in tint, and though it may form papules or even bullæ, never shows the small vesicles or the weeping surface of eczema: its distribution is different, and it is never chronic in its course. Eczema has no resemblance to *psoriasis* except in very old cases of the latter disease, when the scales have disappeared and the locality is obscured.

Prognosis.—Eczema is extremely amenable to treatment, that is to say we scarcely ever see a case in which no improvement can be produced, and still more rarely one which finally resists all therapeutical measures. Moreover, it is scarcely ever dangerous to life. There are, however, exceptions to each of these statements. In the outbreak of acute vesicular and weeping eczema whether primary, or, as far more often happens, occurring in a chronic or nearly cured attack, we can do little or nothing to stop its violence. Abortive treatment is unfortunately rarely successful in any acute disease. Again, in some cases eczema, though improved until very little remains, cannot be driven entirely away and remains in a quiescent state here and there to burst forth again after a longer or shorter interval. Lastly, though it is remarkable how little the general health is affected even by very extensive, troublesome, and long-continued eczema, yet occasionally, in infants, or in aged persons, broken rest or loss of appetite cause wasting and muscular weakness, which may at last end fatally. The only cases of the kind which have occurred to myself were, first, that of an infant which became emaciated, pale, and unable to take the breast, and secondly, an old gentleman considerably over seventy, who after being much relieved by constant tepid baths and other treatment from an almost universal and extremely irritable eczema, sank rapidly and died without any evidence of organic disease. On the other hand, everyone must have seen scores of little children who appeared worn to a skeleton and almost moribund from severe eczema, who nevertheless by treatment or by time, completely recovered. Very general eczema in a person over seventy, especially if complicated with gout or with chronic Bright's disease, should, I think, always suggest a guarded prognosis.

Treatment.—In the first place, it is, I believe, our duty to treat and if possible to cure every case of eczema as quickly, safely, and pleasantly as we can. The supposed danger of driving in eczematous eruption upon internal organs is, I believe, without foundation. It arose partly from theoretical views of the sympathy of organs, partly from the well-known fact that cutaneous hyperæmia diminishes or disappears during acute febrile affections, partly from observing the benefit of counter-irritation of the skin in synovitis or bronchitis, and possibly, as Hebra unkindly suggests, from the difficulty of curing some cases of eczema. In little children, however ill, one may again and again observe that as soon as cutaneous exudation is checked and the irritation subsides, their general health begins to mend. The only caution I would give is to be very careful to ascertain the condition of the heart, the arteries, and the kidneys in aged persons suffering from eczema, lest the treatment or cure of their cutaneous disease should be credited with the fatal result which is really the consequence of degenerated viscera.

Prophylaxis.—The irritants which excite or keep up and renew eczema are chiefly mechanical, thermal, and contact with water. Of mechanical irritants the most important are rough clothing, friction against adjacent parts of skin (intertrigo), prolonged contact with decomposing sweat, and also with dirt and various chemical irritants which are incidental to certain trades. But the most difficult mechanical irritant of all to get rid of is that which is the result of the disease itself. Eczema always itches, and itching is sure to produce scratching. Hence our first attempt is to prevent this by persuasion of an adult patient, by muffling the hands of infants, and by such local applications as will at least relieve the intolerable irritation.

Eczema is common dermatitis and must be treated like other inflammations. Cold, however, is not often practically applicable, powerful as is its influence on inflammation; the surface affected is too extensive, the difficulty of continuously applying adequate cold too great, and the ill-effects of considerable depression of the temperature of the surface too serious, for us to attempt treatment by ice or by cold baths. Moreover, intermittent application of cold by the reaction which ensues proves worse than useless. Eczema in the majority of cases which come before us has passed its acute or subacute stage, and irritation rather than heat is the common symptom. It is, however, always well for patients with eczema to avoid the heat of the sun or exposure to fires or to the heated atmosphere of crowded rooms. The affected parts should not be covered with thick woollen garments, and the patient should lie lightly covered at night; the bedroom should be well ventilated, the temperature kept somewhat low, and much relief is experienced by keeping the feet or arms uncovered except with a thin rag dipped in lotion.

Generally, it is not so much our object directly to subdue the local heat and congestion as to diminish the frequently recurring aggravation of external irritants. With this object, we forbid cold bathing, we forbid the application of either cold or heat, the latter for its immediate, and the former for its consecutive, effects on the circulation of the part. Contact with air is, in many cases, a decided stimulant, and one important use of the various ointments with which the eczematous skin is smeared is to protect it from air. With weeping eczema, we obtain the same end by covering it with wetted rags, or, as is often more efficient, by dusting it with absorbent powders.

But even more important as a cause of irritation is moisture. It is not that mere contact with water is an irritant; probably a slightly alkaline and weak saline solution with a little colloid material, such as gum or size, or the albuminous part of oatmeal, is the least irritating medium with which an inflamed and excoriated skin can come in contact. A continuous bath, even of ordinary water, is a most useful and perfectly safe means of treatment in some cases of very general eczema, with profuse exudation and great irritability. To Hebra is due the merit of proving that patients can be kept continuously in a bath of suitable temperature, not only for hours, but for days—indeed, for an indefinite period, without leaving it for any purpose whatever. I have seen the plan carried out at Vienna, and have more than once adopted it myself. The practical difficulties are obvious, and it is fortunately not often that we need resort to it. But though *continuous* contact with water is by no means irritating, bathing and washing mean *intermittent* wetting of the skin. The change from dry to wet, from higher to lower temperature, and the reverse change on withdrawing the eczematous surface from the bath, the necessary friction of the towel, the saline constituents of most waters, and, most of all, the evaporation which even great care cannot entirely prevent, and which, after careless washing, go on abundantly from the half-dried' surface—these form altogether a most efficient series of irritations.

It is even possible that the frequent and systematic cleansing of the skin from dead epidermis and from sebaceous secretion, which is the result of the artificial condition of extreme cleanliness to which modern civilised society more and more tends, may itself render the skin more susceptible to slight irritants, and certainly with tender skins the use of soaps, of nail brushes, of rough towels, and of flesh gloves, may sometimes aid in exciting dermatitis—a small set-off against the advantage to general health of mind and body to which a clean and active skin undoubtedly conduces.* One most important rule of treatment in eczema, therefore, is that the inflamed parts must not be washed with soap, must not be washed with either hot or very cold water, must not be washed frequently, and must be very carefully dried after washing with soft, dry, and warm towels in order to prevent the "chapping" of the hands which is so common in children during the winter. It is important to take care that they are thoroughly dried on towels which are not already damp. In large schools for poor children, a good plan is to make them dry their hands by dipping and rubbing them in a tub full of bran, instead of upon towels which are sure to be wet for all but the first comers. They should also be given olive oil, or any other neutral fatty compound to rub into the hands after washing, when there is the least appearance of dermatitis. With severe and chronic eczema rimosum of the hands, it is necessary absolutely to forbid washing, and to protect from contact with air or moisture by ointment and a well-fitting kid glove. The best plan in chronic eczema is to advise that once a week, or more frequently as may be thought safe, a complete warm bath and thorough cleansing of the whole surface should be used, taking care to keep the skin immersed from the time of its being first wetted, and to dry it thoroughly when the washing is over. Washing, with unscented, pure yellow soap and lukewarm water, if done seldom and followed by careful drying and inunction, is less injurious to an eczematous skin than more frequent and careless ablution with no soap at all. For the exceedingly irritable skin of the face and hands, it is sometimes desirable, however, absolutely to forbid all contact with water, and cleansing can then be accomplished by friction with dry and stale bread-crumbs. When there is eczema of the scalp the best cleansing agent is white of egg diluted with water.

Poultices are almost, if not always, injurious, and scarcely less so is the modified poultice which is the usual result of the application of water dressing, a piece of lint dipped in water or lotion, and closely covered with oiled silk or gutta percha. The impermeable covering soon raises the temperature, and the result is the combined warmth and moisture of a poultice, most valuable for relaxing tension, promoting suppuration, and relieving deep-seated inflammation, but most injurious in superficial dermatitis.

Local treatment.—We next come to the treatment of eczema by chemical applications. Our object is, first to diminish the hyperæmia and exudation by *astringents;* secondly to diminish irritability and to prevent scratching by *sedatives;* thirdly, to substitute for a chronic and interminable process of inflammation a more directly traumatic, acute, and self-limiting process, or else, it may be, by less stimulus to produce an effect short of this but serving to quicken the natural process of physiological repair. Such agents have received the vague title of *alteratives.*

The most powerful chemical astringent which can conveniently be used is probably lead. (See a paper by Dr Payne in the 'St Thomas's Hospital Reports' for 1878.) Salts of copper, zinc, and iron, nitrate of silver, boracic acid and borax are also efficient astringents. So are galls, tannin, and similar vegetable preparations, though these are less applicable to the skin than to mucous membranes. As local sedatives we may use belladonna, opium, chloroform, hydrocyanic acid, but these are generally unsuitable to eczema on account of its extent and raw, denuded surface, both which characters make absorption too probable for these narcotics to be safe. More efficient as remedies against itching, and free from any but local action, are preparations of zinc, which combine antiphlogistic and antipruriginous qualities. Dilute solutions of carbolic acid, 1 per cent. in water, 1 in 40 in oil, are very useful. Weak tarry preparations are also efficacious, especially in the drier forms of eczema; as diluted oil of juniper or liquor carbonis detergens with vaseline in the proportion of two drachms to an ounce.

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* See an amusing article by Hebra on the dangerous consequences of being overmuch clean, translated in the 'London Medical Record,' March 15, 1877.

For chronic and no longer very irritable eczema more stimulant applications are necessary; sometimes stronger tarry preparations, unguentum picis liquidæ or pyrogallic acid. These are most useful in limited patches of scaly and very chronic dermatitis with much thickening of the skin; those, in fact, which approach most nearly in appearance and pathology to psoriasis.

A still more energetic method is that which was, I believe, introduced by Dr Anderson, of Glasgow—painting the eczematous surface with liquor potassæ. This must be done with much caution, for it gives rise to considerable pain, though in many cases I have found this less complained of than I had expected, but I can bear witness to the efficacy and safety of the treatment when applied tentatively on limited surfaces of old and obstinate eczema, especially of the dry kind. With moist secreting surfaces of unusual obstinacy, I have found more useful the application of a solution of nitrate of silver varying from a scruple to as much as a drachm to the ounce. It must be occasionally painted on, not kept in constant contact, and often proves most efficient as an astringent and a sedative, as well as an alterative.

But more often there is too much active inflammation for us to venture on such treatment, and more generally applicable alteratives are the various preparations of mercury, corrosive sublimate in solution, white precipitate ointment, red oxide ointment, and dilute nitrate of mercury ointment. Mercury in some form is particularly adapted to pustular forms of eczema, and is seldom suitable to those which profusely secrete serum.

Most often, however, the cases of eczema which come before us combine the characters of inflammation, itching, and chronicity, so that for perhaps the majority of cases, at least if we include those of impetigo of the scalp, there is no more useful preparation than such a combination of zinc, lead, and mercury as forms the unguentum metallorum of the Guy's Pharmacopœia. This may be varied by substituting the red oxide for the nitrate of mercury ointment and by varying the proportion of the three constituents; often again lead and zinc act better without mercury, or the carbonate better than the alkaline acetate of lead.

Whatever be the chemical application used it is important to decide whether the vehicle should be watery or oleaginous. An excellent general rule was that of the late Dr Hughes Bennett, of Edinburgh: for dry affections of the skin, use ointments; for moist, use lotions. If an ointment is applied to a profusely secreting eczema, the drug and its vehicle are washed away by free exudation and never reach the subjacent skin. Lotions, on the other hand, have but little power of penetrating the epidermis, and if carefully watched will be seen to run from the surface, which is greasy by its natural sebaceous secretion. With raw surfaces which do not secrete profusely, either lotions or ointments may be appropriately used. Practical considerations teach us that lotions are better suited to diseases of exposed parts like the face and hands, that they are readily applied to young children, that they are more efficiently used by persons confined to bed or by women living indoors than by those who are engaged in active work, that they are more cleanly and pleasant to most people, but that they also give more trouble and demand more time in their application, and lastly, that in the summer, when the skin is frequently covered with sweat, they are particularly grateful and efficient. We must remember that lotions should in most cases be used with exposed skin or with the surface only covered by a thin rag into which the lotion has soaked. If applied in the morning and covered up till night they speedily become water dressings, and probably in less than an hour mere applications of rag with no further therapeutical power. On the whole, therefore, notwithstanding the rule I have quoted above and the fact that eczema is pre-eminently a moist tetter, it will, I think, be found that with the majority of our out-patients, whether private or at the hospital, ointments are practically the more eligible vehicle. It is important to make sure that the lard or other oleaginous material is not in the least rancid, and that it is free from salt. The addition of benzoic acid as now ordered in the British Pharmacopœia makes as good a vehicle in most cases as can be wished. The mineral oils have the advantage of not decomposing, and for some reason ointments made up of vaseline suit certain cases of eczema better than those prepared with animal fats.

Unmedicated oily applications, vaseline, cold cream, olive oil, have in themselves the good effect of protecting from air and of softening rough, harsh skin, inspissated sebum and dried secretions. Glycerine, from its strong affinity with water, is well known to be a direct stimulus to a nerve-trunk. It is, except when dilute, a decided irritant in eczema, and has far from the same soothing effects of cold cream or zinc ointment when applied in cases of intertrigo, chilblains, and eczema solare. In very small quantities, however, it may be added to lotions with the view of securing some of the advantages of an oily preparation.

Quite apart from the ordinary use of a lotion, the whole object of which is to keep the part continually wet, is that of a solution which when painted on is allowed to dry. For this purpose nitrate of silver or other strong astringent solutions may be used. Another useful method is to suspend insoluble powders like oxide of zinc, starch, or sulphate of lime or bismuth in water by help of a little mucilage or tragacanth, without, however, attempting to form a perfect emulsion. The milky liquid is applied freely with a large camel-hair brush or sponge, and is allowed to dry over the weeping surface, and in some cases of irritable and profusely secreting eczema, as also in pemphigus, I have found this the most effective application. Care must be taken not to have too much of the suspended ingredient or hard cakes are apt to form which crack and become painful. In fact, chalk or gypsum shaken up with water and applied like whitewash is sometimes the simplest and pleasantest method. Weak alkaline lotions have often been recommended to relieve the burning pain and irritation of acute eczema, and I have seen them extensively used by Professor Hardy at St Louis. I have, however, generally found that in the very cases of acute weeping eczema in children to which such treatment seems applicable, the parts are so excessively tender that even a 1 per cent. solution of bicarbonate of soda is ill borne, so that in such cases the lead lotion (liq. plumbi subacetatis dilutus of the British Pharmacopœia) is more useful. At all events, if soda is used at all it should be in quantities only just sufficient to react to test paper.

Lastly, we may apply our remedies directly as dry powders. In this way oxide of zinc, chalk, and other fine insoluble powders may be used, and these mineral salts are, I think, better than starch. They dry up discharges, protect from the air, and are often the best applications in cases of intertrigo. On the other hand, they are unsuitable for pustular eczema, where they would form massive and troublesome crusts.

It must, however, be admitted that nothing but experience, insight, and previous knowledge of particular cases will guide one aright in the selection either of appropriate astringents, in the strength of the application, or in the kind of vehicle. Some patients assure one, and I have again and again proved its truth, that they cannot bear any kind of ointment. With others all lotions are apt to produce pustules or even boils. Not unfrequently, especially in the acute stage of eczema, an inert powder or unmedicated vaseline, according as the surface is moist or dry, will do more good than anything else. In all cases we should remember that the ointments are not to be rubbed in, but gently smeared on the skin, and afterwards kept in continual contact by well-adjusted soft linen bandages; that the lotions should never be allowed to get hot, and must be frequently renewed; and that the strength of our applications should be small in the acuter stages, and greater as the case becomes inveterate.

I believe, in accordance with Hebra's teaching, and contrary to that of most English writers, that the majority of cases of eczema can be cured by well-directed local measures of the kind above indicated, but I admit that in the great reforms he established Hebra undervalued the treatment by internal measures, which undoubtedly holds an important, though, I believe, a secondary place.

Diet.—In the acuter stages of eczema, the patient should be put upon almost febrile diet, but should be encouraged to drink freely of any cooling beverage. He should take no stimulants or meat, and eat sparingly, chiefly of bread, milky dishes, green vegetables, and ripe or stewed fruit.

In ordinary chronic eczema no such strict diet is necessary. It is, however, usual to forbid certain articles of food, and I think that the experience of patients shows that, at least in some persons, one or all of these really aggravate the disease, chiefly, I think, by producing thirst, increased irritation of the skin, and scratching. The kinds of food referred to are salt meats of all kinds, including ham and cured fish, cheese, pepper, spices, and other hot condiments. The stronger wines and malt liquors are also usually forbidden, but although in the necessarily generalised treatment of hospital out-patients this is doubtless good advice, I do not know any evidence that the moderate use of malt liquors or wine (with food) does harm in eczema or any other affection of the skin—except in cases where, independently of dermatitis, even moderate stimulants provoke dyspepsia with flushing of the face or symptoms of gout. In many patients, especially those in middle and later life, I am sure that wine or beer with the principal meal of the day helps digestion and certainly does no harm to the eczema, while a little spirit and water at bedtime will help sleep and in that respect prove a useful adjunct to other treatment. Sometimes, however, even weak whisky and water produces heat and discomfort after retiring to bed and must then of course be interdicted. In almost all cases a somewhat free supply of unstimulating diluents should be taken between meals, and a glass of water while dressing of a morning, and again the last thing at night is almost always useful.

Watering places.—In chronic and obstinate diseases like eczema, patients frequently ask whether change of air would do them good. They are usually recommended to go into the country if they live in town, or to go to the seaside if they live in the country, or if they can afford it, to go to Scotland or Switzerland or some other attractive place of resort. The only point on which I can speak with any confidence as to the effects of air and climate upon eczema is that just as it is aggravated by the east winds of an English spring so it is more difficult to cure in the eastern counties of England and Scotland, and is often favourably influenced by removal to the moist and soft air of the western Highlands, of Devonshire or of Ireland. As one sees, however, in so many other diseases, it is the change which does the good, and this is most apparent when the change is from an unfavourable climate. Secondly, there is no doubt that in many cases of the more irritable forms of eczema sea air proves a decided irritant. It is only now and then in chronic and non-pruriginous eczema or in the impetigo of childhood that sea air and even sea bathing do good instead of harm.

With respect to baths generally I have already sufficiently insisted upon the evil effects of frequent contact with water, but there is no doubt that in the very chronic and intractable forms of eczema, saline and sulphurous baths act beneficially, probably like the stimulant and alterative applications above described. When the period has arrived for their use, it is difficult to say, and each case must be judged by the tact and experience of the physician. A single bath may bring back in all its virulence an eczema which had nearly disappeared. Long-standing dryness, thickening of the skin, and absence of excessive irritability are the features which should generally weigh with us in advising or permitting this mode of treatment. The baths best adapted for the purpose are perhaps those of Harrogate.

Internal treatment.—Lastly, we come to the treatment of eczema by drugs, which I regard as less important than that by external applications and by what may be generally called the hygienic treatment of the skin. Still there is no doubt that while we could better dispense with this group of remedies than with the others, we should often fail for want of them, or the success of our treatment would at least be less rapid and complete.

In the acute stage of eczema with profuse exudation and much irritation, it is the practice of the French school to purge freely, and most English physicians adopt the same plan, though perhaps less systematically. Saline laxatives are, I believe, the most useful in these cases. The old-fashioned white mixture of sulphate and carbonate of magnesia taken three times a day, or the pleasanter combination of Epsom salts with carbonate of soda in peppermint or cinnamon water, are useful and popular medicines. A seidlitz powder or a dose of Rochelle salts or Carlsbad salts every morning is suitable for less acute or less extensive cases. Often it is sufficient for the patient to take a draught of Püllna, Friedrichshall, or Hungarian bitter water. Of these three I prefer Friedrichshall, particularly when the eczema occurs in a gouty subject. Sometimes, however, it is less efficient than a seidlitz powder, and occasionally I have found it produce much griping without satisfactory result. In such cases I have often changed it for the Hunjadi Janos with advantage. It is probably a mere fancy, but I have certainly thought that the Hungarian laxative water agrees better with women than with men. Whichever form of laxative is selected it should be taken with a large draught of warm water early in the morning and on an empty stomach. Such a dose should give one or two loose motions after breakfast without griping or subsequent irritability, whereas even larger doses, if undiluted with water or taken with the stomach already full, are more slowly absorbed and produce more frequent and less effectual irritation. In cases of eczema in which the patient has other independent evidence of gout it is well to combine with moderate laxatives the exhibition of a pill containing colchicum and aloes or rhubarb every or every other night. In persons who have lived freely and who are subject to hepatic dyspepsia beside restricted diet both in food and drink and moderate laxatives it is important to prescribe small doses of mercury, either a single grain of blue pill with a little nux vomica and rhubarb before dinner, or two, three, or four grains with an equal quantity of the compound rhubarb pill every other night or twice a week.

In many cases, especially in women affected with eczema, there is considerable anæmia, and then steel must be added to the laxative medicine. There is no better combination for this purpose than that of sulphate of iron given in doses gradually increased from two to ten or even fifteen grains, with half a drachm or more of sulphate of magnesia, five or ten drops of dilute or aromatic sulphuric acid in peppermint, cinnamon, or chloroform water. Along with laxatives it is usual in cases of chronic eczema to prescribe acetate of potash and other diuretics. Their action is, I think, somewhat uncertain, and it is very seldom that there seems to be indication for the employment of such efficient diuretic drugs as digitalis, squill, or the resin of copaiba, but salines, and especially those of potash, have other actions beside that upon the kidneys, and in ordinary cases of eczema with much secretion and extensive inflammation, I believe that citrate of potash or acetate of potash is usefully prescribed, and is perhaps more beneficial as well as more agreeable than the alkaline carbonates.

We have seen above how important a point it is to relieve the itching of eczema, not only for the comfort of the patient, but to secure the physiological rest which the night should bring to all inflammatory processes and also to save him from the serious aggravation of his disease which scratching and rubbing the eczematous parts infallibly causes. Moreover, it is at night (even during sleep) that the irritation increased by the warmth of the bed reaches its maximum, and that the self-control of the patient is weakened or abolished. Beside the various measures above mentioned for securing coolness, protection from the air and such help as local sedatives can give, it is often necessary to call in the aid of internal narcotics. Of these opium and its preparations should generally be avoided. Unless given in large doses they are apt to increase rather than to quell the irritation of the surface; they also check secretion and bind up the bowels. It is therefore better to prescribe chloral hydrate or bromide or the two together.

Chloral should be avoided with old people and with patients who may have disease of the heart or atheromatous arteries. On the other hand, it is extremely well adapted to young children, and I have found a dose of syrup of chloral the most harmless and useful sedative in the case of infantile eczema. The safest plan is to give a moderate dose when the child is put to bed, and repeat it towards midnight, and, if necessary, again towards morning. Fifteen or twenty drops (two to three grains) may be given with perfect safety to a child of six months old; half a drachm twice or even thrice repeated in the night may if necessary be given to a child of twelve or eighteen months; and after infancy, say from two to five or six years old, half a drachm, or for older children a drachm of the syrup may be given at bedtime with safety. Again and again I have seen this treatment followed by the best results. In the first place the child gets rest, and in the morning is ready for food, and all its organs have profited by the natural refreshment of the night. Next, the skin has been free from fresh irritation, and instead of being marked with the little sufferer's nails, is paler and less angry than the night before. All the processes of repair have had opportunity to go on; the habit of pruritus is broken for the time, and the nervous apparatus concerned has escaped from a vicious circle of inflammation, itching, scratching, and increased irritation.

The bromides are unsuitable to infants from their bulk and disagreeable saline taste, but with older children five or ten grains of the bromide of potassium may be sometimes added with advantage to the chloral draught if suitably covered with syrup of lemon or orange. With adults nocturnal irritation is not usually so severe as with children, and a draught of bromide of potassium or ammonium, with or without the addition of chloral hydrate, is usually sufficient when a sedative is required. Fifteen or twenty grains of ammonium bromide, with ten of the potassium salt, and twenty drops of aromatic spirits of ammonium in an ounce of camphor water, forms an effectual and not unpleasant sleeping draught.

In some cases, especially in old persons, neither bromide, nor chloral, nor a combination of them, acts well. Henbane may then be prescribed with advantage, but in doses of not less than a drachm of the tincture either alone or with a little compound tincture of chloroform in camphor water, or I have sometimes with apparent benefit combined the tincture of hyoscyamus with that of hop. Indeed, in old persons where the irritation is not severe, and the want of sleep is rather dependent on general conditions of their age than upon pain or pruritus, two to four drachms of tinctura lupuli is a pleasant form of sedative, and may take the place of the whisky and water which the ascetic habits of the patient may render distasteful, or which habits of the opposite kind may render too agreeable. In the happily unfrequent cases of severe and intractable pruriginous eczema in aged persons we are sometimes compelled to resort to continual doses of opium as the only means of obtaining rest.

The exhibition of antimony has been recommended in the acuter forms of eczema, and, unless I am deceived, has sometimes proved useful in my experience in subduing vascular excitement and hastening the passage of the acute into the chronic stage of the disease.

In many cases of eczema, especially in children, the patient is thin and pale, with poor appetite, and frequent feeble pulse. It is in these cases that a little wine or malt liquor is not only admissible but often extremely useful, and here it is that the exhibition of iron finds its proper place. In the case of anæmic women with constipation I have already mentioned the combination of sulphates in which iron is best prescribed. For children the syrup of the phosphate, the saccharine carbonate, or the citrate of iron and quinine, are all valuable remedies. For infants steel wine is also a popular and valuable remedy, but after eight or ten years old effectual doses are too large to be convenient. In cases of great anæmia in children, especially where there is diarrhœa, no preparation of iron is so useful as the tincture, guarded, if necessary, by twice the number of drops of glycerine, or its taste concealed by a little syrup. It would almost seem as if the astringent quality of the drug had an effect upon the profuse secretion of the eczema. The result at all events is often striking as well as beneficial.

With the exception of iron the group of so-called tonics are not, I believe, generally indicated in the treatment of eczema. Quinine, however, has a very distinct effect in certain cases, so far as I have seen confined to those of infants, children, and persons below adult age, in preventing itching. Half a grain of sulphate of quinine may be given for this purpose to a child a year old an hour before bedtime, a grain if a year older, and as much as five grains to a boy or girl of fifteen. I learnt this effect of quinine from the lamented author of this work, and am glad to see that it has been independently discovered and strongly recommended by Dr Eustace Smith in his work on the 'Diseases of Children.' This writer also recommends guaiacum in the treatment of eczema, especially where there is reason to suspect a disposition to gout.

There remains a drug which in England especially has been very largely and often far too indiscriminately used in the treatment of eczema as of all other diseases of the skin. I refer to arsenic. It is undoubtedly a therapeutical agent of the utmost value in psoriasis, in pemphigus, and in certain other cutaneous diseases to be afterwards described, and no one of experience can doubt its efficacy in certain cases of chronic deforming arthritis, neuralgia, idiopathic anæmia, leuchæmia, and anæmia lymphatica; but like all powerful medicines it is powerful for evil as well as for good. In the acute stages of eczema, in most cases where there is extensive and active inflammation, and in most cases accompanied by severe pruritus arsenic is decidedly injurious. In other cases, however, its success is so marked that in spite of its frequent failures it has never lost a certain reputation in the treatment of eczema. The indications for the exhibition of arsenic are, first, that the eczema must be in a chronic condition—the greatest benefit is obtained in cases which have persisted for years; secondly, the more dry and scaly the surface, the more infiltrated and indurated the skin, the less there is of active inflammation and the less disturbance of the stomach and intestines, the more likely is arsenic to be beneficial. As a rule, children with eczema do not need it, but some of the most striking instances are in very obstinate and long-continued cases in young patients. One especially I remember in which a boy of fourteen, who from five years old had been the subject of what by his own and his mother's testimony was a really unintermitting condition of eczema spreading from time to time with excessive violence from its favourite seats over almost the whole body, but never absent from the scalp, the ears, and the limbs. When I took him into the hospital there was dry scaly eczema of the head, face, and neck, and the hair was very thin. There was eczema rimosum of the ears and axillæ, papular dermatitis of the arms and back, and eczema rubrum madidans of the abdomen, genitals, perinæum, nates, and thighs. The only parts of the whole surface free from the disease were the palms, the soles, and the shoulder. He was thin, worn, and miserable, and the whole skin was so deeply pigmented that he looked like a mulatto, but the urine was perfectly healthy, and he had no other disease than this severe dermatitis. He was carefully treated with zinc and lead ointment and unguentum metallorum as he had been before while an out-patient, but he was also given arsenic in steadily increasing doses from three drops up to fifteen three times a day. Under this treatment the inflammation gradually subsided, and at the end of five weeks it was reduced to a little ordinary eczema of the arms. This also gradually disappeared. Meantime he had also grown into a stout, healthy-looking lad. He has from time to time come to me again with slight return of eczema, chiefly in the scalp and arms, but it has never in the least approached its former severity, and the skin generally, instead of being thick, rough, hard, and infiltrated, with almost entire absence of subcutaneous fat, is now smooth, soft, plump, and elastic, while his head is covered with a thick growth of hair.

In prescribing arsenic the following rules will, I believe, be found useful:—To begin with a small dose, and gradually but steadily increase it until either obvious benefit results, or the physiological action of the drug is shown by itching of the eyes or slight nausea. When these occur the dose should be at once stopped, and then resumed in somewhat smaller proportion, and if necessary again cautiously increased. Secondly, it should always be given either with or immediately after food and sufficiently diluted with water. I know of no advantage in any other form of the drug over Fowler's solution. The arseniate of soda may be given in somewhat larger doses, but is probably converted into the same form during digestion. The hydrochlorate is useful if we wish to combine it with the tincture of steel. Arsenical pills (the Asiatic pills of Vienna) are in every respect less eligible.

Troublesome and difficult to treat as many cases of eczema are, sometimes rebellious to the very treatment which in apparently similar cases has proved effectual, and always liable to relapses which are most trying both to patient and physician, it is nevertheless very rare for us to fail in at least relieving the miseries of an attack, and in a great majority of cases we may be fairly said to cure a disease which, without skilled treatment, would linger on almost indefinitely. Hebra concludes one of the most masterly and original chapters in his great work by saying that he who having once decided upon his plan of treatment, follows it out with patience and determination, will attain his object sooner than he who often changes the measures that he uses. I would venture to add that while keeping steadily in view the broad principles of treatment based upon rational pathology and tested by experience, the most successful practitioner will be he who knows how to vary their application in accordance with the perpetually varying needs of each individual patient.

There are, however, certain practical points in the treatment of local varieties of ordinary eczema which must be briefly mentioned.

Eczema of the ears is one of the commonest local forms of eczema and is sometimes extremely troublesome. Ointments will be found almost always to suit better than lotions—lead, zinc, or equal parts of the two, or in some cases weak carbolic oil, 1 in 40. When extremely moist, powders suspended in thin gum are better than dry powders, which are almost sure to form thick crusts and produce bleeding.

Chronic eczema of the *meatus* may cause deafness by swelling or the accumulation of its products. This must be treated by syringing with soap and water and, if necessary, application of an alkaline wash followed by unguentum plumbi or unguentum metallorum made soft by an equal part of carbolic oil.

Eczema of the *scalp* is complicated by the presence of hair and of sebaceous secretion and is apt to become more or less pustular. The hair should always be kept short, but shaving is unnecessary. Unguentum metallorum is commonly a good application. In the drier form of eczema of the scalp with scarcely any exudation, which is often combined with seborrhœa sicca under the name of pityriasis capitis, tarry applications are most efficient, and none is better than liquor carbonis detergens, either diluted to form a lotion or, as I have found better, with vaseline in the proportion of a drachm or half a drachm to the ounce. Impetigo capitis will be presently considered separately.

Eczema of the *eyelids* and adjacent parts is apt to cause considerable inflammatory œdema which resembles erysipelas, but the colour, undefined edge, and the absence of marked febrile symptoms, together with the almost certain presence of ordinary eczema in other parts, distinguish the two.

Eczema of the *lips* is sometimes confined to that part and has then a peculiar aspect, there being very little serous or purulent secretion, great swelling, deep cracks, thin scabs, and considerable hæmorrhage. When chronic, large thin scales, partly epithelial and partly dry secretion, are formed which have led to its being called psoriasis labialis. The difficulty is to keep the parts from movement. Very mild ointments, vaseline with zinc, yellow oxide of mercury, or honey and borax will be found useful. Deep and painful fissures should be touched with nitrate of silver, either in strong solution or (what is less painful) with a pointed pencil.

Eczema of the *palms* is usually bilateral and confined to these parts or it may persist here after it has disappeared from the rest of the body. As above mentioned, it is often directly dependent upon irritants. Having made sure that the case is not one of syphilis, the first and essential point of treatment is to protect the hand from contact with all other irritants, and especially with soap and water. For this purpose scabs, scales, and crusts should be carefully removed with sweet oil, or if necessary by poulticing. The cleansed surface should then be anointed with unguentum metallorum and thin rags covered with the same ointment should be closely applied to each affected part. A well-fitting thin kid glove should then be worn over the whole; and the dressings should be changed night and morning only. At the end of a week the improvement will generally be striking, or if not it will be due to some neglect of the patient in uncovering his hands or in washing them. If the parts are very irritable, it is better to use diluted white precipitate or yellow oxide ointment, or occasionally unmedicated vaseline will be most effectual of all. In chronic indolent cases, on the other hand, a little of the red oxide ointment will often stimulate most usefully. If there is great accumulation of epidermis it must be removed with soft soap or Hebra's diachylon ointment. Deep and painful fissures should be touched at once with lunar caustic.

Eczema of the matrix of the nail is still more local and residual than eczema of the palms. It is comparatively rare, as a complication of ordinary eczema and a precisely similar inflammation of the matrix of several nails is sometimes seen where there is no other evidence of its eczematous character. The ill-formed nail may be scraped, but its removal is unnecessary and useless. The grooves around it should be carefully anointed with some form of mercurial ointment.

Eczema of the *nipple* has been observed to lead to carcinoma, and if for this reason only should be cured as quickly as possible. It often spreads to the whole breast quite independently of intertrigo of the lower part and is sometimes most difficult to heal. In obstinate cases the application of liquor potassæ has been recommended.

Eczema of the anus, perinæum, and genitals is sometimes confined to the immediate neighbourhood of the rectum. This eczema ani, prurigo podicis of Willan, lichen podicis of Hardy is, as these names imply, most frequently dry and papular and is apt to be intolerably itching ; the irritation is sometimes most severe, especially while in bed, while the disturbance of the rest, and the remarkable effect of mental depression which is common to most of the disorders of this region make it sometimes a truly miserable complaint. French writers describe it as sometimes associated with a profuse and almost paroxysmal discharge of mucus from the rectum. This form of eczema is most common in elderly persons and is often associated with portal congestion, hæmorrhoids and the hepatic dyspepsia described in a previous chapter. In children it most commonly depends upon the presence of thread-worms. Occasionally it is started by fissure of the anus, and disappears when this has been cured by division of the sphincter.

The scrotum and penis are frequently the seat of eczema, most often of the weeping form: eczema vulvæ closely resembles eczema ani in its symptoms and, like it, most frequently affects persons beyond middle life. It is sometimes associated with, and probably dependent upon, diabetes, and sometimes appears clearly due to inflammation, new growths, or degenerative changes in the uterus or bladder. Eczema of the anus, perinæum, or genitals often proves very rebellious and leads to great thickening and induration of the parts affected. Borax lotion or lead ointment, according to the degree of moisture, relieve perhaps more frequently than other applications, but this is one of the forms in which one must be content with tentative measures in each patient. In some obstinate cases a drying lotion of nitrate of silver proves effectual when other means fail.

Intertrigo.—Where this occurs in infants, finely-powdered starch and oxide of zinc or chalk is the best application. In the fold of the nates it is better treated by extreme cleanliness and the application of vaseline or diluted white precipitate ointment. Glycerine to most skins proves an irritant rather than a healer.

Eczema of *the legs* due to *varicose veins* must be treated like varicose ulcers, by elevation and bandaging. An old-fashioned flannel bandage often proves a cheap and efficient method. Martin's elastic bandage often produces the most valuable results, but in wearing it or an elastic stocking care should be taken that the pressure is not too great.

So-called *eczema marginatum* of the thighs is essentially a form of ringworm, and will be described under parasitic diseases.

PUSTULAR DERMATITIS—*Impetigo capitis, Pustular eczema of the scalp*—is one of the most frequent diseases in children. It was known to our forefathers as "scald-head," but has happily become far less common than it was when children's heads were more neglected than at present, and especially when the bad habit prevailed of covering the scalp with caps and linen hoods, indoors as well as out, by night as well as by day. Perhaps the majority of cases are due to the irritation of pediculi capitis, but there remain a large number where no such cause can be found, and where a similar eruption upon the face or other parts establishes its independent character. There is no doubt that we are right pathologically in counting these forms of dermatitis as belonging to eczema. They are superficial and never leave scars, they are often associated with ordinary characteristic eczema of the ears, the limbs, or the trunk, and the same child may be affected at one time with what will be called impetigo of the face or scalp, and at another with eczema of the same parts; or in an infant with ordinary eczema of the scalp the dermatitis will be seen to become more pustular as the hair grows thicker over the head until it has assumed all the characters of the porrigo favosa of older writers. Nor does the fact that this dermatitis of the scalp is often dependent upon dirt, lice, and other irritants prevent our regarding it as true eczema, if the principles laid down on p. 616 are correct. That something beside a traumatic cause, an *irritabile* as well as an *irritans*, is necessary for the production of impetigo, is proved by the fact that some children and most adults may have pediculi capitis for many years, and may even suffer from the irritation, and yet be free from impetigo.

I have already mentioned the best treatment for impetigo of the scalp when associated with ordinary eczema. The children who are the subjects of it are often rosy, plump, and in every way healthy, though here as in other cases it is necessary to judge by the trunk and limbs as well as by the face. If notwithstanding fat cheeks and ruddy complexion, the child is found to have flat shoulders and nates, thin arms and thighs, apparently disproportioned knees, and ill-developed pectoral muscles, his impetigo should be treated not only with *unguentum plumbi et zinci* or *unguentum metallorum*, but also by careful attention to diet, by Gregory's powder, with or without a little grey powder, and when the digestive disorder is corrected, by cod-liver oil.

Impetigo affecting the scalp or face alone without ordinary eczema, and in a healthy child, is happily not difficult of cure. Indeed, apart from the purely pustular secretion, from the eruption being discrete and with a defined margin, and from the absence of severe itching, these typical cases of impetigo are separated from eczema by the fact that they are not prone to recur. Zinc ointment is the popular remedy for the eruption, but its efficacy is much increased by the addition of equal parts of white precipitate ointment or by the substitution of unguentum metallorum. The hair should be cut short, but there is no need to shave it, and the parents may be assured that it will grow all the better afterwards. It is only in extremely rare cases after the inflammation has penetrated to the hair-sacs, owing to a deeper suppuration of the scalp from too strong local irritants, that the hair-sacs are destroyed, and a bald cicatricial patch results. Such an event I have, I think, seen more often from impetigo of the scalp in an adult than in the far commoner cases in children. When, as is usually the case, the scabs are thick and massive they should be removed first by poulticing. In circumscribed cases the bread and water poultice may be used, but where the whole scalp is covered it should be anointed with linseed oil and a large linseed poultice be then applied. For circumscribed and strongly adherent crusts, soft soap or even liquor potassæ may be necessary. Great patience and gentleness should be used in removing the scabs, or the child will suffer considerable pain, and the cure will be retarded.

Impetigo a pediculis.—In all cases of pustular inflammation of the scalp the hair should be carefully searched for pediculi. Equally decisive of the cause is the discovery of the nits, which consist of small triangular cases containing the eggs, made of hard material, in colour and consistence like dried size, adhering to the hairs by one side of the triangle, and visible to the naked eye. The impetigo which results from their presence is produced more by the scratching of the patient than by the irritation of the lice. It affects the back of the scalp chiefly or exclusively, and is attended with great consecutive swelling of the posterior cervical lymph-glands. Indeed occipital impetigo is almost synonymous with impetigo a pediculis. The treatment is decisive and efficient. In bad cases, the whole of the long, tangled and filthy elf-locks should be cut off, and the head washed with soap and water; but in slighter cases it is not necessary to cut the hair at all. The noxious insects are readily destroyed by mercurial washes, but an equally efficient and harmless remedy is the stavesacre ointment (strength ʒij ad ℥j). Common petroleum oil is also a cheap and efficient parasiticide, or if the hair is cut short, as is much the best plan, in hospital practice the white precipitate ointment which cures the disease will also kill the vermin. The egg cases are less easily attacked, and might be sources of future trouble. They must be either combed off, or removed with spirits of wine or cut off, hair and all. The impetigo which results from pediculi will sometimes heal spontaneously as soon as they are removed, but usually unguentum metallorum, or white precipitate ointment, diluted to 1 in 3, hastens recovery.

Contagious porrigo.—This form of impetigo of the scalp has been separately described, and some authors have laid much stress on its distinction both from pustular eczema and from impetigo a pediculis, but I think it must be admitted that no sharp line can be drawn. All impetigo is more or less traumatic and is more or less eczematous, and I would add that all impetigo is more or less contagious. Nor is it in the scalp alone that contagious pus is secreted. The most virulent of all is the pus of a gonorrhœa, but no one can doubt that even it is of varying degrees of activity when we consider the frequency of the urethral inflammation compared with the comparative rarity of gonorrhœal ophthalmia. Leucorrhœa is supposed as a rule to be non-contagious, and no doubt with justice, but the most experienced surgeons admit the possibility of infection from an idiopathic and apparently innocent discharge. Again, the pus of boils is extremely contagious, and is often the source of what is called ecthyma. The pus of scabies, too, is contagious. So also a whitlow may, in children, cause by contagion impetigo of the hand, of the nates, and sometimes of the scalp. Impetigo *a pediculis* often secretes pus of a most actively contagious kind, the proof being not only in the outbreak of similar pustules on other parts of the child's body, especially the fingers and the buttocks, but also in the spread of the disease in children of the same household. Most cases of impetigo are only slightly if at all infectious, whereas in others a whole family or a whole street may be infected from a single case. It is said that the most contagious forms of impetigo are characterised by thick yellow scabs, by a sharp line of demarcation, and by readiness of cure by local means as well as by absence of itching, by restriction to the scalp and face, and by being practically confined to children; but these, after all, are what I should put down as the characters of impetigo generally as distinct from ordinary eczema.

PAPULAR FORMS OF CHRONIC SUPERFICIAL DERMATITIS

(*PIMPLY TETTERS*)

Definition—Relation to common dermatitis and to eczema.
LICHEN—*Its traditional species—Strophulus—Lichen planus ruber.*
PRURIGO—*Pruritus—Prurigo senilis a pediculis—Idiopathic prurigo—Characters and treatment.*

The group of diseases to be now described is far from being as natural and well defined as one could wish. They agree with eczema in being inflammatory, in beginning as papules, in affecting only the epidermis and the papillary layer of the cutis so that they never leave scars, in their essentially chronic course, in the itching rather than pain they produce, and many of them in obstinacy and liability to recur.

But they differ in the following important points:—(1) The inflammation never goes on to the stage of exudation either of serum or of pus; they are all dry tetters. Even when by scratching common inflammatory exudation follows, the pustules or raws thus produced are limited by the cause and do not assume the form of eczema or impetigo. (2) They are much less symmetrical, and rather avoid than choose the favourite places of eczema. Their locality may be said to be undefined, widely diffused, and affecting rather the trunk and outside of the limbs than the eczematous regions.

LICHEN.—Many dermatologists express by this term a papular dermatitis which by subsequent more or less exudation of moisture, or by its symmetry, or its association with previous or subsequent attacks of ordinary eczema, proves itself in my judgment to be more properly papular or abortive eczema. Its pathology, natural history, prognosis, and principles of treatment are precisely those of the drier forms of eczema. It most often affects the arms and legs, and the extensor rather than the flexor aspect.

Lichen circumscriptus vel circinatus is the name given to a peculiar and characteristic form of this papular dermatitis. It occurs upon the trunk, usually between the shoulders, but may spread over great part of the back, or may affect the chest or abdomen. The papules are small, red, and arranged in patches with somewhat well-defined margins. It is not very irritable, and rarely, if ever, ends in ordinary moist eczema. Many cases which have been described under this head are, I am convinced, nothing but papular dermatitis or local eczema depending upon the irritation of decomposing sweat. The locality between the scapulæ and on the front of the chest is just where sweat accumulates; the eruption is most common in summer, and in persons who sweat freely; along with the papules true vesicular sudamina may often be detected. But apart from this it must be admitted that there is a distinct, though somewhat rare, circumscribed papular dermatitis, which from the shape of its patches and from their spreading at the edge while the centre returns to its natural condition reminds one of spots of tinea. Parasitic fungi are apt to occur in the locality and under the conditions named, as in tinea versicolor, but I have never found microscopical evidence of fungus in the affection under consideration.

The areæ of the circles present a yellowish tint, and are sometimes covered by branny desquamation. When several circles combine they form irregular lines, and the eruption has been called *Lichen gyratus* by Biette and Cazenave.

The form described by Wilson and also by Hardy affecting the extensor aspect of the forearm and the back of the hands and running an acute course, should, I venture to think, be regarded as eczema papulatum.

Lichen tropicus.—I have seen only three or four cases of this curious affection, so well known in the East and West Indies under the name of prickly heat. It occurs also in Australia and on the west coast of Africa. Its characters are the sudden appearance of the eruption, its almost universal distribution, and the intense irritation it produces. After once attacking a patient it is apt to return with each hot season, and though usually cured by a temperate climate sometimes comes before us in England. In the cases I have seen the eruption has been entirely papular, with no other lesion but scratch-marks or occasional wheals. The parts most affected were the abdomen, buttocks, and thighs. The face and scalp, the hands and feet, and the genital organs seem to be usually free. One must speak doubtfully about a disease of which the personal experience of an English physician is small, but from its acute character, the absence of moisture, and its uneczematous distribution, we think it better to put it here under papular dermatitis than under eczema. The small red papules are frequently associated with sudamina.

It was once supposed, as with other eruptions of the skin, that driving it in by a cold bath was extremely dangerous, but more than fifty years ago Dr Johnson, who gave a graphic account of it in his own person, justly ridiculed this superstition.

The late Dr Tilbury Fox regarded this as essentially an adenitis of the sweat glands, the direct result of excessive heat and perspiration.

Dr Duhring, of Philadelphia, in his excellent textbook of dermatology, calls it miliaria papulosa on the same theory.

The *Lichen agrius* of Willan is by its acute course and the presence of small vesicles filled with a straw-coloured fluid, clearly a papular eczema.

Lichen pilaris was a name applied by Willan to a familiar condition which is, however, not a dermatitis at all. The hair-sacs of the affected part of the skin become filled up with horny cuticle which forms rough papular projections, hard, pointed, and very characteristic both in appearance and feeling. They do not occur in places where the hair is long, but are almost exclusively confined to the outside of the limbs, over the vastus externus most often, but not uncommonly more or less developed on the outside of both arms and legs, on the buttocks, and the shoulders. This condition is most common in the brawny skin of muscular working men, and may be readily removed by soap and water and friction. Occasionally I have seen it on the limbs of delicate children, more than once in girls of only seven or eight years old.

This affection was described by Devergie under the equally inappropriate term of *Pityriasis pilaris*. It may be better called *keratosis*. Dr Fagge proposed for it the name *Rhinoderma*, from ῥίνη, a file ('Guy's Hospital Reports'), but as he himself says, this term has such obvious disadvantages that he prefers Devergie's title. Willan's *Lichen lividus* is purpura.

Lichen urticatus (Bateman) seems to be nothing but papular erythema combined with urticaria.

The term *Strophulus*,* applied by Willan and Bateman to certain papular eruptions in infants, is now deservedly abandoned. Willan defines lichen as a papular eruption occurring in adults, so that the original distinction between the two diseases was merely one of age. They resemble each other in the anatomical lesion and the subjective symptoms.

Strophulus albidus is not dermatitis but milium, a variety of comedo which will be mentioned in the chapter on affections of the sebaceous glands.

Strophulus intertinctus and *S. confertus* will be called infantile lichen by those who keep to this name. They are papular dermatitis of more or less acute form, and in most cases may fairly be termed eczema.

Strophulus volaticus, with its acute course and slight maculæ following the patches, is a typical erythema papulatum. Bazaine and Hardy are unable to class these papular eruptions of infants among the chronic inflammations which they ascribe to the dartrous diathesis. The former writer places them under it among the scrofulides, the latter among what he ingeniously calls Maladies cutanées accidentales. The *Strophulus prurigineux* of these authors is identical with infantile prurigo, which will be considered hereafter.

Green remarks that "strophulus differs from lichen in no essential particular, a circumstance that might warrant us in discussing the two diseases under one and the same head." ('Compendium of Diseases of the Skin,' 1836, p. 174.) This author points out the difference in age of the patients, the more frequent intermissions of strophulus, and its milder character.

Rayer regarded strophulus as infantile lichen, but Wilson described them separately. Most auhors admit that the papules so closely resemble those of lichen as to appear identical with that disease. They are indeed only modified by the age of the subject.

Lichen scrofulosorum, or as it is more conveniently called, lichen scrofulosus, is a somewhat rare form of eruption which was first accurately described by Hebra. He describes it as consisting of papules arranged in groups with some amount of pigment, and slight desquamation, not itching, and lasting for a long time without change. It is almost always confined to the trunk, and in forty-five out of fifty of his original cases the patients showed swollen lymph-glands, or chronic disease of the bone, or scrofulous ulcers, or, lastly, from a swollen abdomen, were regarded as subjects of tabes mesenterica. On the other hand, in none of the cases was there evidence of phthisis.

Hans von Hebra ('Die Krankhaften Veränderungen der Haut') calls it *scrofuloderma papulosum*. It does not appear under any form of Hardy's scrofulides. It might, however, well be included under those which forms one division of the large group of scrofulides bénignes of Bazin. All Hebra's cases occurred in young men, the youngest patient being fifteen, and the eldest twenty-five. His description has been followed by subsequent German writers, who have added little to the account which

* Strophulus—This name, derived from *στρόφος*, a swaddling band, was apparently first used to describe any skin eruption occurring in an infant. A popular English name is red or white gum, or tooth-rash. These names point to the popular explanation of all cutaneous rashes, and most other affections which occur during teething; but there is, I think, no doubt that "red gum" is only a corruption of another and older term—"red gown," a not inapt description of a child covered with general erythema, and this word gown, meaningless without the prefix, is no doubt nothing but a translation of *Strophulus*.

he gives. I saw two of his cases in Vienna, and can testify that they were not, as has been naturally supposed by some writers, cases of pityriasis scrofulosorum sive tabescentium, xerodermia or dry rough scaly condition of the skin not uncommon in phthisis and other wasting diseases. Kaposi has made sections of the skin, and describes the sebaceous glands as blocked by epidermic plugs, and surrounded by a copious infiltration of the leucocytes, so that according to this excellent observer the disease would be a chronic inflammation of the corium surrounding the sebaceous glands. The late Dr Tilbury Fox transcribed Hebra's account without comment, only stating that the condition is "of infinitely rare occurrence in England." Dr Liveing has met with a few typical cases among poor out-patients, and thinks that the inconspicuous colour of the papules, and the absence of itching, leads to its being often overlooked. I have not seen more than two or three well-marked cases in England; one in a patient of Dr Payne's, a girl aged seven, pale and with swollen glands. From English experience it is more common in children than in adults, and is as common in one sex as in the other. As Dr Fagge suggested in the 'Guy's Hospital Reports,' lichen scrofulosus would probably often be regarded as a form of lichen circumscriptus, or by an earlier generation as strophulus. Its locality, the circumscribed patches, the pale colour of the papules, and the yellowish pigmentation, together with entire absence of itching, are sufficient characters for diagnosis, and seem to justify its recognition as a distinct variety of chronic papular dermatitis.

The treatment consists in the internal administration of cod-liver oil, and is said to be uniformly successful.

Lichen planus.—There is a form of chronic superficial dermatitis which is so distinct from all others that it is well entitled to a separate name. Of all forms of papular dermatitis it recedes furthest from typical Eczema and approaches nearest to Psoriasis or dry tetter, which will be described in the next chapter. It does not appear to have attracted the attention of the older dermatologists and is indeed a somewhat rare disease. It was first described under the title lichen ruber by Hebra* and shortly after from a different point of view by the late Erasmus Wilson† under the more distinctive name which has been generally adopted in this country.

No one who has seen a well-marked example of this affection can doubt the accuracy of Wilson's description; the raised flat patches, their dull glistening surface, deep purple-red colour, slight desquamation, chronic course and resulting pigmentation are very characteristic. Hebra insists upon the genuine papular origin of the affection, on the deep red colour of the papules and their not increasing in size when once formed; but fresh papules appear, so that at last they become confluent and form the raised flat patch which struck Wilson. Hebra gives an elaborate table of the differences between this lichen ruber and lichen scrofulosus, psoriasis, eczema, and pityriasis rubra. It is still a question whether the disease described in Vienna is really the same as lichen planus, but I think there is no doubt that in pathology and the essential points of their natural history the disease is one, although, like many others, not always conforming to the same absolute type. Hillier in 1866 and Neumann more fully in 1869 have described the histology of the disease. The hair-sacs and adjacent sebaceous glands are the chief and apparently earliest seat of infiltration. The opening of the hair-sacs is wide and funnel-shaped, a fact noted by Hebra in his original account. The cells of the rete mucosum contain granules of dark-brown pigment, the natural papillæ are enlarged, the sweat-glands are unaffected, the sebaceous glands atrophied. There is after a time considerable induration of the skin as in other forms of chronic dermatitis.

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* 'Handbuch,' Band i, p. 315, 1860.

† 'Diseases of the Skin,' 6th edition, 1867, p. 190.

The late Dr Fox, Mr Hutchinson, and Dr Liveing, as well as Dr Duhring in America, regard Hebra's and Wilson's disease as unquestionably the same. Mr Wilson himself suggested that the cases described by him were varied examples of the lichen ruber of Hebra, but the latter authority considered the two affections to be distinct, and this was also the opinion of Dr Fagge. Hans von Hebra describes two forms, the first more acute with greater formation of scales, more itching and more generally diffused dermatitis, and also followed by more severe affection of the general health, and the second attack more chronic, never spreading over the entire surface, with only slight irritation and with no injurious effects on the health. This view of their relations is very much what I expressed in a paper in the 'Guy's Hospital Reports' (vol. xxv, p. 254).

Distribution.—Lichen planus may occur upon the extremities or trunk. It has never been observed upon the face or head. Hebra describes it as sometimes affecting the palms and soles, and this statement is confirmed by Wilson and by Hutchinson. The patches are apt to be most marked in parts subject to friction, as the waist and circle of skin pressed on by the garter. Its favourite positions are the extensor surface of the arms and legs, especially the forearm and wrist, and the leg below the knee. In the latter position the colour is more deeply purple than in the former.

It is often symmetrical, but less decidedly so than psoriasis or eczema.

In cases which agree more nearly with Hebra's lichen ruber the papules are of a brighter colour and more generally distributed over the limbs and trunk. The papules and raised patches are not arranged in groups as in most forms of lichen.

Natural history.—Lichen planus is chronic in its development and cure. Hebra's patients were almost all men. In England it has been more often seen in women. It seldom or never attacks children. According to some writers it only occurs in adults, but I have seen a typical case in a girl of thirteen. Writers differ as to the existence of itching. In the cases I have seen it has once or twice been absent, sometimes troublesome but never severe, that is, not comparable to the irritation of eczema, scabies, or prurigo. In extensive cases the nails may be affected. Hebra describes lichen ruber as leading to marasmus and death. The late Dr Fox says that in both forms of the disease the more general and severe of Hebra and the more local of Wilson, the general health is bad. Mr Hutchinson says, that the large majority of patients believed themselves to be in their usual health when it began, but if it persists long and is severe the general health may fail. All the cases I myself have seen were in persons of average health, some of them in robust health. No internal organ is affected nor are there any symptoms of general disturbance, at least in ordinary cases. The occasional implication of the nails is, I think, more rare than chronic eczema affecting the nails.

Lichen ruber is undoubtedly often associated with the white patches on the tongue and cheeks which have been described under the varied titles of ichthyosis linguæ, psoriasis linguæ, tylosis, keratosis, and leucoplakia. This, as is well known, frequently occurs independently of lichen or any other cutaneous disease. The association was noted in two of his cases by Mr Hutchinson ('Lectures on Clinical Surgery,' vol. i, pp. 211, 213), and I have seen it more than once.

Diagnosis.—This affection is distinguished from eczema by its never forming either vesicular or raw surfaces, by its avoidance of the face and ears, its distribution generally, and the comparatively slight amount of itching. In some cases it undoubtedly approaches very closely to psoriasis, especially to inveterate cases of the latter disease which have become generally diffused and have lost much of their characteristic appearance. Mr Hutchinson would recognise transition cases, and, indeed, proposes to name lichen planus "lichen-psoriasis." But difficult as the diagnosis occasionally is, the distribution, the character of the scales, and the persistence of papules will in most cases sufficiently distinguish lichen planus from psoriasis.

A much more important distinction is between lichen planus and syphilis, for which I have often seen it mistaken. The colour, the frequent absence of itching, and the somewhat irregular distribution, lead to this mistake, which is apt to be confirmed if white patches are found on the tongue or cheeks. This leucoplakia, however, is not any proof of syphilis. The colour of lichen ruber is more purple and less brown than that of syphilide. The freedom of the face and scalp, the absolute uniformity of all the lesions, and their persistence, unchanged during long periods of time, ought to ensure a correct diagnosis, but the absence of indurated lymph-glands and of other signs of the constitutional disease may sometimes need to be ascertained.

Lichen planus does not tend to cure, it continues indefinitely, may spread extensively, and, as I have above stated, will in certain severe cases affect the bodily health most seriously.

Treatment.—The treatment adopted both in Germany and England is the administration of arsenic. Most writers speak of this as specific and certain in its effects, but some of considerable experience find it occasionally fail, and I have certainly found it slower in yielding to the remedy than average cases of psoriasis. Locally, tar ointments or some of the milder preparations, oils, or ointments, which will be described under psoriasis as substitutes for tar, are important aids in treatment. In obstinate cases pyrogallic acid is worth trying.

PRURIGO.—Prurigo, "the disease attended with pruritus or itching," was a term formerly very loosely applied and is still somewhat difficult to define. Willan described it as a papular eruption in which the papules are of the same colour as the skin and accompanied by itching. Its "species" are *P. mitis* and *P. formicans*, which are merely more or less severe cases of the same affection, and *P. senilis*, characterised by the age of the patients and the difficulty of cure. Bateman thinks that pediculi are not unfrequently generated when prurigo senilis is present, thus putting the cause for the effect, since it is now well ascertained that at least a great majority of cases of prurigo senilis are the direct cause of pediculi corporis.

Willan and Bateman also mention *Prurigo pubis*, which they rightly ascribe to the presence of pediculi, and *P. præputii* and *urethralis*, which are the only sympathetic pruritus. Lastly, their species, *prurigo podicis* and *P. pudendi*, correspond to the drier and more papular and indurated forms of the irritable local dermatitis which was described in the last chapter as eczema ani and eczema genitalium.

1. *Pruritus.*—Prurigo, a papular inflammation of the skin, is distinct from pruritus or subjective sensation of itching without any local lesion. Pruritus accompanies not only prurigo but eczema, scabies, and the desquamative stage of many exanthems. It is the constant result of pediculi; it may be produced by jaundice, and it is also the result of the various atrophic changes which take place in the senile skin. These, which have been well described by Neumann, include the wasting and ultimate disappearance of the papillæ, and it is probable that the process gives rise to senile pruritus.

2. *Prurigo senilis a pediculis.*—This is a well-characterised and common disease known as prurigo senilis, phthiriasis, or prurigo pedicularis. It is

a papular dermatitis of definite clinical characters dependent on the irritation of body lice, and is only seen in elderly persons. It is a good example of the combination of two conditions—the excitant and the predisposing cause, the *irritans* and the *irritabile*—to form a constant pathological and clinical result.

Phthiriasis is not a sufficient title, for children may be swarming with vermin, and may suffer from urticaria or ecthyma as the result, but are never affected with this form of prurigo, and "prurigo senilis" is not enough unless we recognise the exciting cause of the disease. The papules are large and separate, not spreading over wide surfaces as in eczema, nor collected in more or less rounded patches as in lichen circumscriptus, nor coalescing as in lichen planus. Moreover, they are much larger than in eczematous dermatitis, and flat rather than pointed, less red, and more persistent. But what is most characteristic is that before long each of them is capped by a little black crust of dried-up blood, the result of scratching.

Beside these papules, the disease is marked by extensive series of scratch marks following the curves which are described by the right or left hand respectively, working from the shoulder. The irritation of scratching not only causes excoriation and hæmorrhage, but sometimes produces wheals like those of urticaria and raw surfaces which may be properly called traumatic eczema. Both these effects, however, are most often absent, but prurigo senilis never lasts long without the whole surface between the papules becoming more or less deeply pigmented until in some cases the affected parts are as dark as the skin of a mulatto.

The *distribution* of prurigo senilis is as characteristic as its anatomy. It occupies the shoulders, back, and loins, the papules usually stopping abruptly at the waist or the sacral region, and sometimes not spreading below the scapulæ. They may appear over the upper arms, but rarely below the elbow and never on the hand. They are numerous on the flanks and in severe cases may cover the whole chest and abdomen. Even the thighs may share in the disease; but even in the most extensive cases it is generally found that the outlying parts are rather the seat of ordinary dermatitis produced by scratching than of the true papules of the disease. Prurigo senilis never affects the face.

The itching is most severe; like all pruritus, worst at night and when warm, while the absence of pain and tenderness leads to more reckless scratching than in any other disease. It is the consequence, however, and not the cause of the papules, for we can distinguish the latter from the traumatic dermatitis set up by the former.

The exciting cause of the disease can be found when carefully looked for, especially in the plaits of the underlinen about the neck and waist. It is important to remember that pediculi corporis may exist in old men and women of apparently scrupulous cleanliness. The whole facies of the disease is so well marked that it can scarcely escape recognition. It affects both sexes. I have not seen typical cases in persons as young as fifty.

The *treatment* is simple and effectual when the disease is once recognised. The most effectual parasiticide is the white precipitate ointment, and if only applied to the shoulders I believe no harm will ensue; even when used somewhat freely, as is apt to occur in out-patient practice, I have only twice known it cause salivation. One of these was in a very fat and burly man of about fifty-four years of age, who having extensive prurigo continued to rub in the ointment beyond the time intended.

Inunction, such as we use with grey ointment in cases of syphilis, is quite unnecessary; it is enough for the parts to be smeared over.

3. There remain certain forms of disease which must be regarded as prurigo, and which are quite independent of pediculi. They agree,

however, with prurigo senilis in the anatomical character of the papules and in the excessive itching to which they give rise. One form alone is the prurigo of Hebra, of which striking description I must refer the reader to the Sydenham Society's translation, vol. ii, page 258. He admits milder cases which correspond to the prurigo mitis and formicans of Willan, but would separate them broadly from the severe form which is congenital and incurable. I saw cases of this Hebra's prurigo as it has been called at Vienna, and venture to think that their characters have been somewhat over-described, if not exaggerated, by the great dermatologist. At all events cases have been described, both in America and in England, which agree with it in all essential particulars, and I have myself seen cases in London which would make an uninterrupted series connecting the worst of those in Vienna with the slightest forms of infantile prurigo.* I think, therefore, that we may fairly include these affections under a common name, using such adjectives as mitis, agrius, congenital, infantile, inveterate, to denote the varieties which we find in practice.

Anatomy.—The papules of prurigo are at first scarcely distinguishable in colour, and, as Hebra says, are felt rather than seen. They are not closely set, and do not appear in patches, they produce great itching which causes black spots and scratch-marks as in prurigo pedicularis. The skin between is more or less pigmented, and is generally covered with a fine branny desquamation. In course of time it becomes thick and indurated and in many cases there is traumatic eczema, often of a pustular kind. In severe cases, inflammatory enlargement of the lymph-glands occurs both in the groin and the axillæ. The distribution of the eruptions is generally on the trunk and limbs. The face is almost always free, and also the flexures of the joints, palms, and soles. It is generally most severe on the back, chest, and abdomen, on the buttocks, the shoulders and upper arms, and it is generally worse on the lower than on the upper extremities, and worst of all below the knee.

Prurigo begins in early life and either disappears during childhood, or if present in an adult, has persisted from that period. It is generally worse in winter and has hence been named prurigo hyemalis. The latest age at which I have known it begin was fourteen, in a lad whom I saw on several occasions up to the age of eighteen. It began on his legs, and affected the whole surface except the head, palms, soles and flexures. There were a few spots on the cheeks and neck, on the hands and penis; the trunk was moderately affected, the buttocks and thighs more so, and the arms and legs most of all. There were severe buboes, and he was thin and wasted. He improved greatly under treatment, but the disease returned from time to time.

In twenty consecutive cases observed, the ages were, under twelve months two, between two and five eight, between five and ten one, between ten and fifteen five, between twenty and forty-five three. It is, as I have observed it, more common in men and boys than in females. In chronic cases the skin is often much thickened and pigmented. According to Mr Hutchinson ('Lectures on Clinical Surgery,' vol. i, p. 15) prurigo in children is often the result of varicella. Dr Duhring described winter prurigo in 1874 as pruritus hyemalis, an undescribed form of pruritus. Mr Hutchinson also describes relapses in prurigo chiefly affecting adolescence, and recurring every summer, principally on the face and arms.

A great many cases which were formerly described as prurigo should be called papular dermatitis from the irritation of pediculi (which in infants never cause the characteristic appearance of prurigo) or of bugs, or of friction of flannel next the skin, or papular erythema, or urticaria. Lichen circumscriptus, papular eczema, and even congenital syphilis have been mistaken for prurigo.

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* See, on this point, a paper by Mr Morrant Baker, 'Internat. Med. Congr. 1881,' vol. iii, p. 177, and the discussion which followed.

The treatment of prurigo, even in its most typical and severe forms, is far from being as hopeless as Hebra supposed. Frequent warm baths and assiduous inunction, together with arsenic internally in steadily increasing doses, with cod-liver oil and good feeding, will restore inveterate cases to health and comfort. It is, however, almost certain to return, and probably more than once, and must be kept at bay for years before it finally disappears. The slighter forms of true prurigo in infants and children are very much aggravated by scratching, and the first point is to prevent this by hydrocyanic lotion or other local anodyne, and by sedatives at night as described under eczema (p. 631). In some cases quinine appears to be almost a specific, both for the irritation and the disease.

PITYRIASIS RUBRA

(*EXFOLIATIVE DERMATITIS*)

Name—History—Account by Devergie, Wilson, Hutchinson, Baxter—Course, Symptoms, and Anatomy—Histology—Prognosis—Diagnosis—Treatment.

The word "Pityriasis," meaning, as its etymology implies, a branny or furfuraceous desquamation of the skin, is conveniently used to describe that pathological condition, but no one *disease* is properly entitled to the name.

The species defined by Bateman as *Pityriasis capitis* is in most cases *Seborrhœa sicca,* an affection of the sebaceous glands of the scalp, or it may be slight local dermatitis (*Eczema capitis*) due, as he remarks, to want of cleanliness and removable by soap and water, but apt, if neglected, to degenerate into "Porrigo," that is to become pustular eczema or impetigo of the scalp.

Pityriasis versicolor, now known as tinea versicolor, is a parasitic disease.

Pityriasis nigra, described by Willan as occurring in children born in India, was not identified by Bateman, nor I believe by anyone else. A case of Alibert's which Devergie calls "pityriasis nigra with prurigo," was apparently *Prurigo pedicularis* with pigmentation and leucodermia.

Bateman's fourth species, *Pityriasis rubra,* "resembling psoriasis diffusa," is a stage in the involution of eczema. The case of pityriasis rubra described by Cazenave seems to have been *Tinea versicolor,* with more irritation than usual.

But this same term, "pityriasis rubra," was unluckily applied by Devergie in 1854 to a very remarkable form of superficial dermatitis which certainly deserves a special name. It is probably identical with Alibert's "Herpes squamosus." Hebra in 1860 thought himself bound to follow Devergie's nomenclature, and his authority has made it generally accepted. Wilson's proposed names of "Pityriasis foliacea rubra" or "Eczema foliaceum" (1867), or the better title, "Exfoliative dermatitis" (1870), have not displaced the original term. "Universal exfoliative or desquamative" dermatitis is perhaps the title that would most clearly express its characters.

Devergie* placed it next to eczema from its great resemblance in form to that affection. He describes the disease as beginning with an erythematous redness, usually on the chest or flexor surface of the limbs and spreading rapidly with a well-defined margin, deep colour, abundant scales, and more or less thin serous discharge. It covers the whole body, is very obstinate, lasting for months, and occasionally proves fatal by exhaustion and diarrhœa. As a rule, however, patients slowly recover. Relapses are frequent. Devergie admits the difficulty of distinguishing this new disease from eczema, and bases the diagnosis on the following points:—It is of a deep red colour, it has sharply marked borders, it may affect the whole skin; the skin, and even the subcutaneous fascia, are thickened, less itching, more burning than eczema, its secretion is thin, and does not stiffen linen; the scales are abundant, readily detached, and present from the first no red moist points (*état ponctué*) under the scales.

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* 'Traité Practique des Maladies de la Peau,' p. 263.

In the 'Glasgow Medical Journal' for January, 1858, p. 421, Dr McGhie recorded a case of "pityriasis rubra acuta" which he rightly regarded as one of Devergie's disease. This was, I believe, the first published in this country, and preceded Hebra's cases. The same patient's condition was described by Professor Gairdner ('British Medical Journal,' March 13th, 1875, p. 359) seventeen years later. Among the early cases may be mentioned one by Dr Wilks in the 'Guy's Hospital Reports' for 1861, which he called "general dermatitis;" the eruption was universal, red, dry, with abundant desquamation. Another was recorded by the late Dr Hillier ('Handbook of Skin Diseases,' p. 101) in 1864, and another by Dr Fagge in the 'Guy's Hospital Reports' for 1876, vol. xiii.

Some authors regard it as essentially Eczema squamosum, and one of Mr Wilson's titles is "Eczema exfoliatum." Dr Liveing agrees with Wilson and Fagge in looking on pityriasis rubra as only a peculiar form of eczema. Mr Hutchinson ('Lectures on Clinical Surgery,' Part I) would separate pityriasis rubra from eczema, and regard it as the type of a group of affections which differ in anatomy, but agree in being universal, in resisting treatment and in often proving fatal. This would include *Pemphigus foliaceus* with certain cases of psoriasis and lichen.

The late Dr Baxter published a valuable paper on this disease under the title of "General exfoliative dermatitis" ('British Medical Journal,' July 19th, 1879). He considers the affection as clinically the result of a universal inflammation, and as arising by the general diffusion of either eczema, psoriasis, lichen, or pemphigus. The objections to this view are that eczema may be nearly if not completely universal, and that for long periods together, without losing its characteristic features and without endangering the health. The same appears to be true of *Lichen planus*, if we accept Hebra's descriptions of universal chronic *Lichen ruber;* for this he carefully distinguishes from pityriasis rubra. *Pemphigus foliaceus* is seldom if ever universal, and differs markedly, as will be seen hereafter, from pityriasis rubra. That the whole skin may be occupied by a scaly eruption without interference with health is proved by many cases of ichthyosis.

Auspitz, who is followed by Hans von Hebra, separates pityriasis rubra from the inflammatory diseases and places it among affections of the epidermis (keratonoses) as *keratolysis.*

Origin, course, and characters.—Pityriasis rubra may undoubtedly arise from eczema or psoriasis or probably any other form of superficial dermatitis, including erythema, impetigo, and traumatic dermatitis, but it most often arises without previous cutaneous lesion, beginning with itching and erythema. It rapidly spreads over the trunk and limbs, but in an irregular fashion, unlike the gradual and methodical extension of eczema or psoriasis. Finally, it affects the whole of the cutaneous surface, including the scalp, the palms, and the soles. The skin is of a full deep red colour, not thickened and indurated as in chronic eczema, covered with profuse and abundant scales, which are large, thin, and usually detached, unlike those of psoriasis or of syphilis or the branny desquamation which follows eczema or the disappearance of the exanthems. They are apt to form successive undulating ridges which Wilson compared to those of the "ribbed sea sand;" they are exceedingly abundant, so that the patient's bed is filled with them by the peck. In most cases the surface is absolutely dry; occasionally there may be a slight inflammatory exudation, especially in the flexures where the inflamed skin is apt to crack. This exudation has not the stiffening property which marks that of eczema. There is more or less pyrexia and general disturbance of health, especially at the onset. If, as is most frequently the case, the disease becomes chronic and inveterate, albuminuria is occasionally observed, and the appetite and health begin to fail. The irritation varies in different cases; it is usually considerable and sometimes almost as intense as in eczema so as seriously to interfere with sleep.

Histology.—In a case of a year's standing examined after death by Hans von Hebra, the whole of the cutis was filled with leucocytes; in another case which had lasted several years all signs of active inflammation had disappeared, the Malpighian layer was thin and its cells shrunken, the papillæ atrophied and the deep layer of cutis transformed into thick bundles of elastic fibres with abundant pigment; the glands had also suffered atrophy. In chronic cases the hair may be lost.

General exfoliative dermatitis is common to both sexes and to all ages. Though more frequent in the latter periods of life it is not unknown in children.

Diagnosis.—Pityriasis rubra is distinguished from *eczema* by its abundant scales, by the absence of visible moisture, and by its not showing predilection for the ears, face and flexures of joints; from *psoriasis* by the thin, loose scales, by its not specially affecting the elbows and knees; from *pemphigus foliaceus* by the scales not being preceded by bullæ; from all these forms of superficial dermatitis by its being universal and uniform in distribution, and by the severe symptoms which usually accompany it.

Prognosis.—This is much graver than that of ordinary dermatitis, eczema, psoriasis, lichen, purigo, or pemphigus. For not only is it difficult to cure but it sometimes ends in death, especially in elderly people. The presence of albumen is a bad sign, though not a fatal one. Emaciation is still more serious, depending as it usually does on loss of appetite or sleeplessness or diarrhœa. Yet the disease is not, as Hebra supposed, incurable; nor is it by any means constantly fatal. Since his book was written cases of recovery have occurred at Vienna. In forty which I collected from various sources, recovery ensued in fifteen cases, improvement in several more, and death only in eight. In these cases the fatal event was caused by bedsores and exhaustion, by lobular pneumonia, by acute pneumonia, or by bronchitis. In other cases marasmus ensues and diarrhœa ends the disease. It often persists almost uninfluenced by treatment for an indefinite period.

Treatment.—Locally the best applications are those which have been recommended in the drier forms of eczema—weak carbolic oil, lead and zinc ointment, liquor carbonis detergens with vaseline (ʒj ad ℥j) freely and frequently applied. Warm baths are not counterindicated and usually give relief, but if too warm they lead to irritation afterwards, and the effect on the pulse must be carefully watched. Arsenic has not the power it possesses with psoriasis and with chronic eczema. It is best given in small doses combined with steel. Bark and mineral acids are often useful. Milk and farinaceous diet appears to suit best, and cod-liver oil should be taken if it does not interfere with other food. Good red wine or sometimes porter I have seen decidedly beneficial. In one obstinate case in an otherwise healthy old gentleman whom I saw with Dr Ford Anderson, complete recovery followed six weeks' sojourn at Strathpeffer in Ross-shire.

Pityriasis rosea of Gibert, and *P. circinnata et marginata* of Vidal, will be described in a following chapter.

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[*] I may be allowed to refer to a paper in the 'Guy's Hospital Reports,' series 3, vol. xxv, in which forty cases of pityriasis rubra are collected, and its nature and symptoms discussed.

PSORIASIS

(*DRY OR SCALY TETTER*)

Frequency—Name—Anatomy and pathology—Course—Symptoms—Distribution—Ætiology—Varieties: guttate form, inveterate form—Relation to pityriasis rubra, to eczema, to syphilis—Prognosis—Treatment.

Excluding scabies and syphilis by far the most common cutaneous disease is eczema; next comes acne, and then psoriasis. Like the affections hitherto described it is a chronic superficial dermatitis, and like them has been described as a dartreuse or herpetic affection. It stands, however, at the opposite extreme from typical idiopathic vesicular eczema, with which it offers points of contrast rather than of resemblance.

An old and good name for psoriasis was dry tetter. The Greek term signifies the condition of *psora* or itching and has no bearing on the present signification of the term. Certain forms of psoriasis were formerly known as *lepra*, which from its etymology, "the scaly disease," would be more appropriate, but the confusion with leprosy is decisive against the word. The Greek term *alphos*, referring to its white scales, was revived by Erasmus Wilson, but without general acceptance.

Anatomy.—Psoriasis is an extremely well-marked and characteristic form of disease. It begins as papules, which rapidly increase in size and form flat patches. From the beginning the scales can be seen upon the papules, and by the time they are as large as a pea the scales form conspicuous white spots. They are large, perfectly dry, strongly coherent, and not easily separable from the skin; they have also the characteristic white silvery lustre due to the abundance of air which is included between the layers of horny epidermis. When the scales are removed the surface on which they rest is seen to be red, shining, and dry, but the injection is not that of acute hyperæmia, and either stops at the edge of the scaly patch or only extends very slightly beyond it.

Histology.—The earlier dermatologists of the present century, Gustav Simon, and even Hebra, were unable to prove, what they recognised as probable, that psoriasis is essentially a form of dermatitis. As with eczema and most other cutaneous affections the characteristic appearance disappears after death. By the better methods of modern histology Vatten and Neumann first established the existence of abundant cellular infiltration of the papillæ of the corium extending along the tracks of blood-vessels in its deeper layers. They also ascertained that the papillæ are enlarged to ten or twelve times their natural size and this papillary hypertrophy is present from the first, not only, as in eczema, in the later stages. The scales of psoriasis, like those of pityriasis rubra, consist almost absolutely of keratin—unmixed with fibrin and leucocytes as in chronic eczema and syphilis, or with sebum as in pityriasis capitis.

Local evolution.—Psoriasis is no less characteristic in the regions it affects than in its anatomical lesion. Its favourite spots are over the olecranon process of the ulna and over the patella, ligamentum patellæ and tubercle of the tibia. In fact it is remarkable how very rarely these spots will be found free even in the most chronic and varied forms of the disease. Here it begins and here it almost always remains. From these points it spreads downwards over the extensor surface of the forearm and on the shin and calf, not, however, by a continuous extension as is the case with eczema, but by the development of separate patches with well-defined margins, which as they increase in size become confluent with the originally diseased surface. The whole upper and lower extremities may be covered with such patches, which by their coalescence form large spaces, but there will always be found more or less extensive islands of healthy skin between the diseased parts, and these will have a concave, while the scaly patches have a constantly convex, outline. On the back or chest the same process is seen on a larger scale. As the raised, red, and scaly edge of the eruption advances, the inner parts which were first affected lose their scales and return more or less incompletely to a healthy condition, so that by this progressive spread and involution of the disease the scattered scaly patches in which it began gradually give place to extensive surfaces of almost normal appearance bounded by sinuous lines of red and scaly skin made up by the intersecting segments of many circles. *Psoriasis gyrata* was the technical term applied to this stage.

After psoriasis has lasted for some time its colour begins to acquire a deeper and brownish tint. It no longer disappears completely upon pressure, that is to say, pigmentation has been added to hyperæmia. In inveterate cases this becomes very characteristic, the colour being of a deep brown, sometimes almost mulatto tint. When the disease has been cured, when the scales are removed, the hyperæmia has subsided, and the finger cannot feel anything but healthy skin, dark pigment-blotches remain to attest the nature of the recent malady. They always disappear in time, but, especially in old persons, their disappearance is slow. It may be said that next to syphilis, psoriasis produces pigmentation more quickly than any other form of dermatitis, and the depth of pigment may be as great as in the most chronic cases of eczema or of prurigo senilis. In this as in other respects psoriasis resembles lichen planus and differs from pityriasis rubra.

Course.—Psoriasis is never acute. Even when it develops rapidly it is unaccompanied by the ordinary symptoms of inflammation and never causes constitutional disturbance. Often a patch on each elbow, or on the elbows and knees, may appear and remain for years before it shows signs of spreading. When it has become extensively diffused and passed through the centrifugal process above described, it will, if untreated, enter upon a very chronic and almost interminable course, the skin being habitually thick, harsh, and dry, and the general aspect resembling that of some of the forms of dry scaly chronic eczema in old persons.

Of all skin affections, psoriasis is most prone to *recur*, more so even than eczema. It is very rare for a single outbreak to occur. Sometimes when the eruption has only just disappeared under treatment, a fresh attack comes on, and the very means which will almost infallibly cure it when developed are often powerless to prevent its return.

Notwithstanding its etymology, itching is comparatively unimportant as a symptom of psoriasis; at least it ranks much below that of eczema, scabies, and prurigo. In many instances there is no irritation at all, in most it is slight, but in a few it is sufficiently troublesome to demand special treatment. It is still more rare for the affected parts to smart or to feel hot and tender. Though pathologically it is an inflammation, it is the most chronic, cold, and uninflammatory of all the inflammations of the skin.

It produces no constitutional effects, and persons subject to it are entirely free from special liability to any other disease. The digestive, urinary, and other functions are carried on as usual unaffected by the condition of the skin.

Distribution.—Psoriasis is of all diseases the most symmetrical, not more so, it is true, than typical forms of eczema, but its range is so much more restricted and its varieties so unimportant, that while typical eczema does not include more than perhaps three fourths of the whole number of eczematous cases, we seldom meet with one of psoriasis which deviates from the characteristic type. As above stated, its favourite or practically its constant seat is upon the two elbows and the two knees, next it is common over the whole extensor surfaces of the extremities, specially the forearm, the front and outside of the thigh, and the calf. Even when most extensive it shuns with scarcely an exception the bend of the elbow and the popliteal space. It not unfrequently extends from the forearm to the back of the hand, and from the leg to the dorsum of the foot, and occasionally may cover the fingers and even affect the nails. Psoriasis unguium is known by the excessive and unsightly thickening of the nail, and by the absence of soreness and suppuration of the matrix. It sometimes occurs when the rest of the fingers or toes is free from the disease, but almost always spots of psoriasis will be found on the elbows or knees, or the patient has previously suffered from the disease. The only other affections of the nails which at all resemble it are eczema unguium above described, onymycosis, to be mentioned under ringworm, and whitlow.

Psoriasis very rarely affects the palms or the soles, and never, I believe, unless other parts of the body are previously the seat of the disease. What used to be described as psoriasis palmaris was probably either eczema squamosum or scaly syphilis. When present the scales of psoriasis are comparatively small, but the patches keep their well-marked edge. There is little or no disposition to form cracks, and the soreness and irritation of eczema of the palms or soles is absent.

Next to the extensor surfaces of the limbs psoriasis is most common on the trunk. The shoulders, back, and loins, the sacral and gluteal regions, are very commonly its seat, somewhat less so the chest and abdomen. Indeed, I have never seen psoriasis of the abdomen which did not affect other parts of the trunk, and it is very rare to find psoriasis of the trunk when the limbs are completely free. The genital organs are occasionally the seat of psoriasis which has usually spread from the abdomen or the thighs, but this is far less frequent than eczema of the same parts, and what used to be called psoriasis scroti is really syphilis squamosa. The face and head are less frequently attacked by psoriasis than the trunk, far less frequently than the limbs, but with so common a disease cases frequently occur in which the red scaly patches appear upon the neck, the cheeks, the forehead, and the scalp. The scales are usually smaller upon the face, the whole aspect less characteristic and apt to be further confused by slight ordinary dermatitis, but the presence of unmistakable psoriasis on the limbs or trunk prevents any mistake in diagnosis. On the scalp the closeness of the hairs prevents the formation of large scales, and the sebaceous secretion gives them a greasy consistence and a yellowish tint. *Psoriasis capilitii* is a not unfrequent affection, and broadly distinguished from eczema and impetigo capitis, from syphiloderma and seborrhœa sicca or pityriasis capitis. It is always dry, the scales are coherent, the hair does not fall out, and it is always coincident with existing or previous psoriasis of other parts.

It is doubtful whether psoriasis ever affects the mucous membranes.

Psoriasis labiorum has been described already as a form of eczema. Psoriasis linguæ is *leucoplakia* or white patches on the tongue, distinct from syphilis and often antecedent to epithelial cancer: so far as I have observed when it is coincident with an affection of the skin, that affection is lichen planus. I do not of course deny the observations of other dermatologists as to the occasional coexistence of these patches with ordinary psoriasis: the only question is whether the coincidence is accidental or not.

Ætiology.—The cause or causes of psoriasis are absolutely unknown. By French writers it is generally ascribed to a *dartrous* diathesis, and by those who go still further into speculation a dartrous is distinguished from an *arthritic* psoriasis. In England it is very commonly regarded as *gouty;* by others, however, it is considered, especially in children, as a *scrofulous* disease, and I believe there is as much evidence for the one hypothesis as the other. Dr Yandell regards a great deal of psoriasis as well as of eczema as *malarial* in origin, and in the last century psoriasis was by most physicians considered as undoubtedly *scorbutic.* Some writers have speculated on the possible connection of psoriasis with *leprosy,* and would have us regard it as the expiring and gradually mitigated manifestation in modern times of the scaly leprosy, white as snow, which is described in the Old Testament. The remarkable centrifugal progress above described naturally suggests the idea of a parasitic vegetable growth. We may confidently assert that no fungus is present, and although it has been asserted that *bacteria* may be found in the affected skin when the scales have been removed, their presence, which is certainly not universal, must be regarded as purely accidental, and would neither explain the course and spread of the disease nor help in its treatment.

It would take far too long to refute the various hypotheses above mentioned. I have attempted to deal with them elsewhere.*

With respect to gout, however, it must be remembered first that the word, both in Germany and even in England, is often applied upon very insufficient evidence. In fact those who use it would sometimes not even imply the presence of urate of soda in the joints. Moreover, the diagnosis of gout is always acceptable to an Englishman of the middle class. On the other hand, it is quite true that few families of rank in this country are free from unmistakable gout in some of their members. But psoriasis is comparatively rare in private practice as compared with that of hospitals. Very few of those who have unmistakable podagra are liable to psoriasis, and psoriasis is as common in Scotland, Germany, and America, where gout is rare, as in England, where it is frequent. If we determine that every disease must have for cause some condition already known, it will be easy to find one in the list I have given above for every case of psoriasis, but I venture to think that such a practice hinders the progress of knowledge of the real causes of disease, interferes with rational and successful treatment, and leads to acquiescence in superficial statements and arguments which is fatal to medicine either as a science or an art.

Psoriasis occurs equally in both sexes and at all ages above infancy; it becomes more common from the age of six or seven up to puberty, and the first attack usually falls in childhood or early adult life. It may, however, begin after fifty and even in old age.

Varieties.—As I have above stated, psoriasis compared with eczema is singularly uniform in its anatomy and natural history. The description above given applies to ninety-nine out of a hundred cases, of course with individual variations, but less than those of even such typical diseases as typhus and variola. There is, in fact, only one variety which demands notice; in children the ordinary form is frequently seen, but more commonly the early spots of *Psoriasis guttata* as above described never grow into the nummular form, and the large patches of gyrate psoriasis are decidedly rare under puberty. This would scarcely be worth mentioning in itself, but the spots are also remarkable for having little or no red border. They produce absolutely no irritation and—as is common with other diseases affecting children—the local distribution is less rigidly marked than in adults. The guttæ are frequently seen on the face, and they are perhaps more abundant on the trunk than on the limbs. Dr Liveing thinks, moreover, that guttate psoriasis occurs in children who are scrofulous, *i.e.* pale and thin. I have, however, often seen it in those who are robust, and certainly in most cases there is no chronic enlargement of the lymph-glands, no caries, no chronic synovitis, and no evidence of tubercle.

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* 'Guy's Hospital Reports,' 3rd Series, vol. xxv, pp. 220, 224, 237, 242.

When psoriasis has lasted for many years and has spread over the greater part of the surface, it loses much of its characteristic appearance, the scales are less abundantly formed, the margins are less definite, and the whole skin becomes thickened and indurated, so that it often requires careful investigation and a knowledge of the earlier stages of the affection to distinguish this *psoriasis inveterata* from the dry and chronic eczema described in a former chapter.

Again, there is no question that psoriasis may pass into, or be supplanted by, the dry scaly and universal dermatitis described in the last chapter as pityriasis rubra. As I have there stated, the late Dr Baxter thought any dermatitis, eczema, psoriasis, lichen, or pemphigus might, if sufficiently extensive, assume the characters of that remarkable disease. In a paper I have referred to elsewhere ('Guy's Hospital Reports,' vol. xxv, p. 266), I have given reasons against accepting this hypothesis, but I have myself had a remarkable case in which a woman who herself had been in St Thomas's Hospital under Dr Payne with ordinary psoriasis of the elbows and knees, and whose daughter was a patient of my own also with psoriasis, came under my care with marked and typical pityriasis rubra. It is very probable that some at least of the cases of general psoriasis, described by Hardy as very rare, would have been recognised by Devergie as pityriasis rubra.

One may admit that eczema and psoriasis, which in so many ways are allied by points of contrast, have connecting links on the one hand, with pityriasis rubra or universal exfoliative dermatitis, and on the other with lichen planus, which, as we saw, sometimes so closely resembles psoriasis, while by its relation to ordinary forms of lichen it has affinities with papular eczema. We draw lines as nearly as we can in accordance with the broad demarcations of pathology and natural history, but here as in other departments of medicine it would be pedantry to deny that there are transition forms which it is difficult or may be impossible to classify.

The important question, however, of the diagnosis between psoriasis and the scaly forms of syphilis is one which rests on the absolute distinction of cause and one which is of the utmost practical importance. The locality and symmetry, the character of the scales, the colour, the presence of itching, the uniformity of the lesion, and the absence of other signs of syphilitic disease are the points to be attended to. The last, however, may deceive, for a man with psoriasis may acquire syphilis, as he may scabies, and I have notes of three or four cases in which true psoriasis and a secondary syphilitic eruption existed in the same patient, ran independent courses, and were cured by different treatment.

Prognosis.—Psoriasis if left to itself lasts for an indefinite time, though almost always getting better and worse at intervals. It never interferes with the health or affects other organs than the skin. After being cured it is of all diseases most apt to return.

Treatment.—The external treatment of psoriasis consists in inunctions of some preparation of tar. Nothing is so effectual as the unguentum picis liquidæ of the Pharmacopœia well rubbed in at night and allowed to stay on while the patient sleeps in a special suit of underclothing; it may then be washed off in the morning, to be reapplied at night. When the scales are very thick and indurated it may be well to precede this application by hot baths and soft soap. Where the smell and colour of tar are objected to, useful though less efficient substitutes may be found in the liquor carbonis detergens made into an ointment with vaseline, two drachms to an ounce, or in the huile de Cade, oleum rusti, &c. Another plan of obtaining the same result is to apply a spirituous solution of tar or the liquor carbonis detergens diluted with water. Goa powder and the chrysophanic acid which it contains are powerful cutaneous stimulants and have been often used with success in the treatment of psoriasis. They occasion, however, with many patients considerable pain, and stain the skin and linen unpleasantly, so that I believe most practitioners have, like myself, given up the use of this once vaunted cure. In cases where tar is inadmissible a much better substitute is pyrogallic acid half a drachm to an ounce of benzoated lard or benzoline, the strength being increased with caution. For rapidity of cure with freedom from unpleasant smell this is perhaps the most eligible of all applications.

Beside the local treatment it is almost always desirable, after the scales have been thus removed, to put the patient upon a course of arsenic. It is usual to prescribe it in a bitter effusion, but I think it will be found to agree quite as well, and to be more constantly taken, if merely diluted with water or flavoured with syrup, cinnamon, or peppermint. It should always be taken at or immediately after a meal. Three, four, or five drops in an ounce of water three times a day is the dose to begin with, and it may be increased to ten or beyond. If properly diluted, and taken with food, even full doses very rarely cause pain, sickness, or diarrhœa. The first sign of the physiological limit being reached is usually the irritation and slight injection of the conjunctiva. As soon as the patient feels his eyes begin to itch he should be instructed to leave off his medicine for a couple of days, and then resume it in slightly smaller doses. He has then reached what is for him his full therapeutical benefit. I have several times cured psoriasis by arsenic without any external application whatever, and, again, have several times cured it by local treatment without internal medication, but in most cases the cure will be hastened by the application of tar, and will be rendered more permanent by the administration of arsenic.

In anæmic persons especially, it may be desirable to administer steel. We may then combine the liq. arsen. hydrochlor. with the liq. fer. perchl. In other cases Fowler's solution is the best. It is the fashion to administer Pearson's solution (the arseniate of soda) in preference to children, but there is little doubt that it is absorbed in exactly the same form. When arsenic disagrees it should not be hastily given up, but the dose should be diminished until unpleasant effects no longer follow, or we may sometimes prevent them by adding a few drops of laudanum or a little compound tincture of camphor to the dose. When the patient suffers from gastritis and sore eyes, five drops of arsenic, three, or even two will probably be sufficient to cure the psoriasis; when a patient can take ten without discomfort it may be that fifteen will be needful to cure his psoriasis.

Children take arsenic very well. When they are pale and thin and ill-nourished, cod-liver oil is often a useful coadjutant. In the guttate form, most common under puberty, local treatment is often scarcely necessary. Purgatives and diuretics are quite unnecessary, and I have never seen colchicum required in the same way as it undoubtedly is in the treatment of certain irritable and probably gouty forms of eczema.

PEMPHIGUS

(*BLADDER-TETTER*)

Names and definition—Anatomy—Histology—Local distribution—Age and sex—Question of an acute form of pemphigus—Diagnosis—Prognosis—Pemphigus malignus—Pemphigus foliaceus—Hutchinson's cases—Serpiginous form—Chiropompholyx—Pemphigus (*vel herpes*) *gestationis—Hydroa of Bazin—Frequency—Treatment.*

Nomenclature.—We now come to a form of superficial dematitis which is rare compared with eczema or psoriasis. Although not less remarkable than these in its anatomical characters, its course and natural history are far less characteristic, its pathology more obscure, and its origin entirely unknown. It has been called by two names, *pemphigus* and *pompholyx*, but of these terms, which, like lepra and psoriasis, were made separate genera by Willan and his disciples, there is no need to retain more than one.

The names *pompholyx* and *pomphus* seem to have been originally applied to what we now call "wheals;" *pemphix* meant a bulla, and pemphigus was applied to a supposed "febris bullosa" of doubtful nature. Bateman practically admits only one bulbous disease, a chronic superficial dermatitis, characterised by blebs; it might be called "bladder-tetter."

It was by Willan associated with erysipelas, a striking example of the results of following an anatomical, or we may add any exclusive, basis of classification for so complex conditions as diseases. He had previously united it with vesicular diseases, but distinguished the two orders in consequence of the criticism of Tilesias, of Leipsic ("über die flectenartigen Ausschläge," in Martin's 'Paradoxien,' 1801).

Anatomy.—The bullæ of pemphigus begin with a scarcely demonstrable papular stage; the first lesion seen is usually a small transparent vesicle which rapidly increases to the size of a pea or larger. These bullæ are sometimes seated on perfectly natural skin; sometimes, however, they are surrounded by a rose-coloured injected ring, but this is narrow. They are never found upon an actively inflamed or swollen surface. They may burst when not bigger than a pea or a marble, but on the other hand will sometimes increase to the size of a billiard ball or more. They are usually tense and hemispherical, occasionally oval. There may be either a single bleb or several of various sizes irregularly scattered over the same region, and when in such groups the intervening skin is often injected of a rose colour. Each bulla, however, forms separately, and it is very rare for two to run together. The contained liquid is transparent, and gives the bulla a pearly appearance. When removed by pricking, it is thin, watery, colourless, not coagulating, becoming opalescent or turbid on heating, and showing a few leucocytes under the microscope. After a time, however, it often becomes turbid from increase of the inflammatory corpuscles, and before the bulla bursts the contents may be opaque and yellow, in fact almost purulent. They do not, however, acquire the thick creamy character of pure pus and always begin as serous and not purulent cavities. Threads of fibrin also appear not unfrequently before the rupture of the vesicle. Still more common is an admixture of blood, which gives a pinkish aspect to the bulla. After it has burst, fresh secretion soon ceases, the ruptured cuticle is either torn off or adheres to the exudation, and the lymph, whether serous, puriform, or coagulated, dries up into a thin yellow crust which may be more or less stained by hæmoglobin. This soon falls off and leaves a smooth, healthy surface, with scarcely any desquamation; but some passive injection remains, and with this may be mingled more or less pigmentation, so that the circular patches, of sizes varying from a sixpence to a florin, remain for some time as characteristic evidence of pemphigus.

Histology.—The inflammatory exudation of pemphigus takes place in the Malpighian layer of the epidermis. The cells of this layer are first drawn out into bands by the accumulating serum, so that in the early stage each vesicle consists of a series of loculi, just as is the case with the traumatic bullæ of a burn, first described by Biesiadecki; but this stage is shorter and less marked than in the case of traumatic bullæ and much less than in the vesicles of smallpox. No scars are ever left, and only moderate pigmentation.

Distribution.—Pemphigus differs from eczema and from psoriasis in being *unsymmetrical*, and having no definite local predilection. The bullæ appear sometimes individually upon distant parts of the body, succeeding one another indefinitely (*pompholyx diutina*); more often two or three up to half a dozen are observed in an irregular patch; and isolated bullæ, or one or two other patches follow in other parts of the surface. Occasionally the trunk and limbs may be so covered that scarcely any region can be said to be entirely free, yet even then the lesions show no preference for one part over another. I do not know any part of the surface on which I have not seen pemphigus. On the trunk and limbs it is most frequent, on the face scarcely less so; the genital organs are sometimes affected, the abdomen and thighs, the ears, the hands and feet; even the palms and soles and the matrix of the nails may occasionally be the seat of pemphigus, the hairy scalp least frequently of all. It is said that bullæ have been observed inside the mouth and on the conjunctiva, but I have not seen this myself.

Age and sex.—Pemphigus, though belonging to the rarer diseases of the skin, may be seen in patients of almost any age. It is commonest in children, decidedly infrequent in adults, but sometimes observed in elderly patients, when it is apt to assume its more severe characters. It is said to be somewhat commoner in women than in men. Among twenty-one consecutive cases of my own, six occurred in men and fifteen in women. Two occurred in children between one and five years of age, ten between five and ten years only eight between eleven and fifty, and one in an old woman of seventy.

Acute pemphigus.—Hebra discusses the existence of acute or febrile pemphigus (*febris pemphigodes*), and like Bateman before him, concludes that when urticaria, erysipelas, rupia and other forms of syphilis, and herpes iris are excluded, there is no such disease. The late Dr Sparks, however, in 'Quain's Dictionary,' says that the existence of such cases is now certain. Dr Southey has recorded a case ('Clin. Soc. Trans.,' viii, 1875), Dr Payne one with table of temperatures ('St Thos. Hosp. Rep.,' vol. xii) and Dr Duckworth another ('St Barth. Hosp. Reports,' vol. xx). The latter occurred in a man of fifty-four, suffering from Bright's disease, and he died on the ninth day, but the event was probably not due to the eruption on the skin, and if not cut short by death, this might have proved chronic. Moreover, it is possible that this as well as other cases might be interpreted as bullous erythema, though it would doubtless be difficult to maintain in every instance the distinction.

Diagnosis of pemphigus—The bullæ of this disease are so characteristic that it cannot be overlooked, and cannot be mistaken for eczema, lichen, psoriasis, or any other of the forms of superficial dermatitis already described. But all bullous eruptions are not pemphigus.

1. The blisters may have been produced designedly or accidentally by local irritants, especially by scalding water, or by cantharides. The traumatic bullæ which follow extreme heat are of two kinds—true inflammatory products containing lymph, chemically and histologically identical with that of pemphigus; and bladders filled with gas, which have been formed by the lymph of the living skin being turned into vapour and expanded by heat. The latter condition was long ago described by Hilton as the result of burns and scalds: but I believe it is of rare occurrence. Its purely physical nature is proved by the fact which he observed, and which I have myself verified, that it is possible to produce it in the skin after death. If a hot iron be held close to the surface, the cuticle rises in a blister like that produced by the sun on a painted board, and on pricking it no liquid is found within. This is strikingly seen in a negro's skin, when the white cuticle is raised from the dark rete mucosum beneath.

Factitious inflammatory bullæ are usually seen on the arms, and in doubtful cases the glistening scales of the elytra of the Spanish fly may be distinguished by a lens.

2. Scabies may be accompanied by bullæ, especially in children. I figured an example of this in the face of a little boy, whose appearance closely simulated that of pemphigus ('Guy's Hospital Reports,' 3rd series, vol. xxii, pl. i).

3. Syphilitic eruptions in the later stage of the disease are often bullous. Usually the exudation becomes purulent and the resulting crusts are massive, dark from blood-pigment, and more or less conical, forming the condition described as "rupia," and leaving a superficial ulcer when they fall off, with considerable pigmentation and final cicatrisation. In cases of congenital syphilis, however, bullæ exactly like those of pemphigus may be observed; so that *pemphigus neonatorum* is probably always syphilitic. Besides other signs of the congenital disease, the appearance of the bullæ upon the palms and soles is a character which is diagnostic.

4. More difficult of distinction from true pemphigus are the bullæ of certain forms of erythema, to be presently described as herpes iris and erythema bullosum (*infra*, p. 670). Their locality, their symmetry, and their acute or subacute course, are the chief marks which distinguish these erythematous eruptions from true pemphigus.

Prognosis.—German authorities speak doubtfully of the prognosis in this disease, and depend chiefly upon local applications for its treatment. In children it is rarely fatal (excluding of course so-called syphilitic pemphigus), and under suitable internal treatment it is in most cases quickly curable. But in old persons it is apt to spread very widely. Sleeplessness and loss of appetite follow, and death may result. This is most to be feared when there is chronic renal disease present, but I have seen it occur independently of this complication.

Such cases have been made a distinct variety, *pemphigus malignus vel cachecticus.* The bullæ are very numerous, are never tightly filled with serum, but look flaccid and rupture early. There is little effort at healing, and extensive raw patches cause much pain and distress, combined sometimes with more or less itching. The exudation is frequently hæmorrhagic and sometimes fibrinous, or, as the German writers call it, "croupous." As in other severe and extensive forms of dermatitis, there is sometimes albumen in the urine.

These serious cases, though rare except in old age, may occur at any age; in children, bullæ after bursting are sometimes succeeded by gangrene, and this *pemphigus gangrænosus* has also been separately described. It has no doubt been frequently confused with what used to be called rupia escharotica and pemphigus neomatorum, that is to say, with a syphilitic bullous eruption. But there is no question that true gangrenous pemphigus does occur. In a little boy, aged four, who died of it in Guy's Hospital, we found, post-mortem, all the viscera perfectly normal.

Ætiology.—The cause of pemphigus is absolutely unknown, although, as in other cases, teething, gastric irritation, excess in diet, irritability of system, mental affections, anxiety, fatigue, amenorrhœa, exposure to cold, and residence in damp situations have been confidently stated as, every one of them, causes of the disease. According to Alibert, the lymphatic temperament predisposes to pemphigus ; which probably means what is true, that it is more common in children than adults.

It is now well established that pemphigus is never contagious. Hebra relates one remarkable case of heredity.

Pemphigus foliaceus.—Cazenave described a remarkable form of cutaneous disease under this title, which has since been recognised by Hebra and other dermatologists. It is rare, and I have myself only seen two well-marked cases, one at Vienna, one in Guy's Hospital.

The patients are usually adult women. The blebs appear at first like those of ordinary pemphigus, but they never become tense and pearly in appearance. They rupture early and form thin, dirty-white laminæ, which continue to exude a scanty secretion. The aspect of the affected skin has been likened to that of flaky pie-crust, to birch-bark, and to dead leaves—whence the specific name.

Beside the anatomy, the distribution of this form of pemphigus is remarkable in being more or less universal. On this ground and its malignancy the late Dr E. B. Baxter associated it with pityriasis rubra (*cf. supra*, p. 648).

Its course is very slow and there is no disposition to recovery. Indeed, it is doubtful whether any genuine case of pemphigus foliaceus has ended favourably. Drugs have little or no influence upon it, and after a protracted illness the patients die emaciated, or are carried off by some intercurrent disease.

Pemphigus uterinus, hystericus v. pruriginosus—herpes gestationis v. impetigiformis is a remarkable and rare affection which has been named as a species of pemphigus, of herpes or of erythema, or has been included under the title "Hydroa." Its pathological alliance appears to be with the form of erythema which depends on ovarian irritation, but the existence of bullæ makes it convenient, if only for purposes of diagnosis, to consider it along with pemphigus.

It occurs only in women during pregnancy. The bullæ, vesicles, and vesiculo-pustules appear in abundant crops over the trunk, and often on the face and limbs also. Pruritus is marked. There is more or less constitutional disturbance and sometimes the temperature rises high. The clinical aspect is therefore serious and occasionally alarming. But I believe the result is always favourable. The disease is cured by delivery.

I have seen one instance—and others are on record—in which this remarkable form of pemphigus or bullous erythema appeared again and again in successive pregnancies.

Cases of "herpes gestationis" have been carefully described since Chausit and Hardy by Dr Liveing, Dr Bulkley and other observers in this country, on the Continent, and in America. The general features are very uniform, and there is no doubt of the reality and distinctness of the disease , but its true nature, pathological relations, and prevention or treatment are still obscure.

Hutchinson's bullous disease of hands and feet.—A curious form of bulbous eruption, which may be at least provisionally called pemphigus, was lately shown by Mr Hutchinson at the Pathological Society, and deserves the name of the hand-and-mouth disease, for beside the bullæ on the trunk and limbs there is severe inflammation of the hands with loss of nails, and also inflamed mucous membrane of the mouth and tongue. I saw a case exhibited at the Dermatological Society in the present year, 1885, which was recognised by Mr Hutchinson as of the same character as his own case. There was unmistakable pemphigus here, and both the loss of nails and sore mouth have been described by Hebra, so that I have no doubt he would include it under pemphigus.

Moreover, the same combination of dermatitis was described by Rayer as complicating pemphigus.

Serpiginous pemphigus is a rare form which arises only in chronic cases. The bullæ, which are small, are placed on the red advancing border of a considerable space of skin, which was formerly the seat of bullæ. When first seen in this latter stage it, like the hand-and-mouth disease just described, might well puzzle an observer. A well-marked case of pemphigus serpignosus, which began as an ordinary case and recurred, and each time was cured by arsenic, is given by Dr Fagge in his 'Catalogue of the Models of Cutaneous Disease in the Museum of Guy's Hospital,' p. 92.

Chiro-pompholyx.—This affection also was described by Mr Hutchinson. It is chiefly confined to the hands and feet, is symmetrical, affects the nails, is recurrent, and the bullæ are small without dermatitis around them. Dr Robinson, of New York, and Dr Liveing have described similar cases. It affects the palm and sides of the fingers as well as the dorsum of the hand. Before rupture, the small bullæ, or large vesicles under the thick skin of the fingers, are described as like sago grains. There seems no doubt that this disease is quite distinct from the affection of the sweat-glands, described by the late Dr Fox as dysidrosis, but I have not seen enough of it to form an opinion worth expressing as to its real nature.

Hydroa.—I ought, in conclusion, to state that the group of eruptions named hydroa by Bazin is not, in my judgment, a natural one, either clinically or pathologically. Of the three species described by him, the first, or vesicular hydroa, would seem by its localization on the back of the hands and wrists, and on the front of the knees, as well as by its acute but sometimes recurrent course, to be clearly a form of erythema. Other cases are identical with the curious affection long known as herpes iris, which will be referred to in the chapter on erythema. The bullous form of hydroa probably includes the pruriginous pemphigus or herpes gestationis above described, and the bullous form of erythema multiforme. Some cases, again, which have been described as hydroa have turned out to be iodide rashes. (See on this subject an elaborate paper by the late Dr T. Fox in the Philadelphia 'Archives of Dermatology' for 1880, p. 16.)

Frequency of pemphigus generally.—Hebra, writing of twenty years' experience in the enormous General Hospital of Vienna, as well as in his large private practice, could reckon about 200 cases. He estimates that, excluding infants, one case of pemphigus occurred in 10,000 cases of illness generally. But we must remember that the hospital statistics apply to all medical as well as surgical diseases, whereas his own practice was exclusively dermatological. He found in thirty years' statistics at the General Hospital, that there were ten cases of pemphigus in men for rather more than three in women, excluding pemphigus foliaceus. In a report to the American Dermatological Association in 1881, only twenty cases occur in 11,000.

Treatment.—Here we have cause for congratulation on improved knowledge. In Joseph Franks' work on diseases of the skin, he stated that the best treatment of pemphigus is to leave it alone. Hebra proved the uselessness of diuretics and purgatives, tonics and quinine, mineral acids, Rayer's vinegar, and Carlsbad waters. The older English physicians recommended venesection or leeches with antiphlogistic regimen, but with a caution to pursue the plan guardedly, which probably meant not to pursue it at all. They go on to recommend acids and bark. In the first edition of Wilson's work, he writes, "When there is reason to believe that the eruption is an effort on the part of nature to determine to the surface a morbid disposition, I should strongly recommend the employment of mustard baths to the entire surface of the skin, or a stimulating liniment of some kind, such as that of croton oil in the proportion of a drachm to an ounce of olive oil, to be well rubbed into the parts of the skin." Hardy says that "le traitement général du pemphigus est encore à trouver." The late Dr Tilbury Fox recommended tonics: chlorate of potash, good food, and above all quinine, which he preferred to arsenic. At the present time, however, I believe that all English physicians are agreed that arsenic is as much a specific remedy for pemphigus as for psoriasis. No doubt it occasionally fails even in ordinary cases, which can certainly be said of mercury in syphilis, and no one pretends that it will cure gangrenous pemphigus, or pemphigus foliaceus, or bad cases of extensive pemphigus in aged patients; but in nine tenths of the cases of pemphigus occurring in children and young adults, I fully agree with the statement published in the 'Medical Times and Gazette' for February, 1854, by Mr Hutchinson, that arsenic may be esteemed almost a specific remedy, even in severe cases. Since this he has adopted still more favourable opinions. The author of these volumes, the late Dr Fagge, as well as Dr Habershon, Dr Hillier, Dr Gee, and many others support the same opinion (see the 4th of Mr Hutchinson's 'Lectures on Clinical Surgery:' "Can Arsenic cure Pemphigus?"). The drug should be administered on the same principles and with the same determination as recommended in psoriasis.

Occasionally, however, we meet with cases on which the drug seems to have no effect. After perseverance with varied doses and varied forms of administration we are obliged to abandon arsenic—not because of its disagreeing, that can always be met by diminishing the dose—but because the disease is unchecked. The best treatment then is tincture of steel. Others have found quinine, guaiacum, or cod-liver oil succeed when other drugs have failed.

Locally no applications are very useful; whatever is most soothing is best, either zinc ointment, or oxide of zinc in powder, or what I have found more often pleasant and effectual, oxide of zinc with finely powdered chalk or gypsum suspended in water, and applied with a large soft brush. In very extensive and severe cases, continuous baths have proved useful. In true gangrenous pemphigus of children, when syphilis has been excluded, brandy and strong broths, or raw meat, with chlorate of potash internally, is the best treatment, and commonly proves successful. But even in infants oneminim drops of Fowler's solution should, I think, always be administered. In bad cases, especially in aged patients, attended with restlessness and distress, opium is a most valuable remedy, but unfortunately its use is forbidden, or much circumscribed by the not infrequent presence of albuminuria. When this is absent it has a most valuable effect. So far as I know, neither arsenic nor any other drug is of any benefit in pemphigus foliaceus.

SCABIES

(*ITCH*)

Importance of the disease—Its nature—The acarus—The superficial dermatitis it produces—The distribution of the parasite and of the inflammation—Diagnosis—Treatment.

Scabies, though once scarcely accounted worthy of a place in nosology, and though without the interest of danger, is really one of the most important diseases from a scientific point of view ; for, if this were the place to enter fully into its history and pathology, we should find, I think, that it illustrates the whole progress of scientific medicine,—the ancient method which still survives of inventing explanations instead of investigating circumstances, the fallacy of ascribing results to dyscrasiæ of which the existence has never been proved, the survival of doctrines in pathology which have long been exploded in physiology, the importance of apparently useless knowledge, the bearing of pure sciences like zoology upon practical therapeutics, the nature of inflammation and the relation between an irritant and an irritable tissue, the eradiation of sensations, the pathology of pruritus, and the importance of a patient's nails in the production of cutaneous lesions. Finally, scabies is the typical example of a disease which is now as fully known as it is perhaps possible for us to know any disease ; of which we know the pathology and the cause, of which we can explain the symptoms, which we can diagnose with certainty, which the boasted *vis medicatrix naturæ* is utterly powerless to affect, but which we can cure by definite, simple, and rational means, quickly, safely, and completely.

Scabies, like the affections which have hitherto occupied us, is a superficial dermatitis; in the character of its lesions it may even be called a common superficial dermatitis, for they do not essentially differ from those which will be produced by any common mechanical or chemical irritant of sufficient energy, and are exactly comparable in their anatomy to the vesicles of eczema, the papules of lichen or prurigo, the bullæ of pemphigus, and the pustules of impetigo. Hebra, therefore, since as we have seen he called all common superficial dermatitis of traumatic origin "eczema," logically describes scabies as a form of eczema. But, as I have explained in a previous page (p. 617), eczema is not a mere traumatic dermatitis, and scabies must be separated from all other diseases because its cause, its prognosis, and, above all, its treatment, are totally different. We may define it as a superficial dermatitis of various degrees of severity, but always accompanied with intense pruritus, which results from the invasion of the skin by a parasitic acarus and from the scratching which ensues.

The itch-mite.—The living cause of this disease is the female itch-mite known as the *sarcoptes hominis*, formerly known as the *acarus scabiei*, belonging to the acarine division of the class Arachnida. It has four pairs of legs (which at once distinguish it from parasitic insects) and is clothed in a chitinous integument furnished with abundant bristles. The male acari, which are much the smaller in size and fewer in numbers, live upon the surface of the body, but do not burrow. The female after impregnation digs her way into the integument, forming a straight, curved, or sinuous *cuniculus* (mite-burrow, *Milbengang, sillon* or "run") which is visible to the naked eye as a slightly raised ridge, with a dark depression at one end (the entrance clogged with dirt) and a slight papule or small vesicle at the other, where the parasite lies. A lens of low power shows these characters more perfectly, but it is comparatively rare to see the runs perfectly well developed, for they are injured by the inflammation set up, by the patient's scratching, by friction and by dirt. When fresh they are best seen in the soft skin between the fingers and on the ulnar and palmar side of the wrist, still better when present in the skin of the prepuce and penis, or in that of the mammary gland in women. In children their locality is less certain, and they are much less easily found. With quick eyesight and a little dexterity the burrow may be laid open with a needle from the entrance to its blind extremity, and the acarus, a minute white grain just visible to the naked eye, extracted. It generally clings to the point of the needle, but a microscopic slide with a drop of water, glycerin, or liquor potassæ, should be ready to receive it. The needle should be sharp, stout, and not too long or elastic; some prefer the broader needle of the oculist. Another plan is to excise the parasite, burrow and all, by means of a sharp pair of scissors curved on the flat. The winding passage can then be demonstrated, with the black granular fæces of its inhabitant, and often with a row of oval eggs in chitinous shells, which are laid one by one as the acarus bores deeper into the skin. Sometimes the needle or scissors may fail to secure the parasite, but will prove its presence by that of one or more of its ova.

The dermatitis.—The presence of the acarus produces irritation which in most cases is intense, equal to that of the most irritable eczema or the worst kinds of prurigo; but often it is comparatively slight, only annoying the patient after he is warm in bed, when the skin is more vascular, the papillæ more sensitive, and possibly the acarus more lively, while the patient has nothing to divert his attention from his own sensations. The degree of inflammation also varies extremely, and cannot always be explained by the more or less severe scratching of the patient. As above stated, there is usually a vesicle formed at the end of each run, but beside these, large vesicles, bullæ, and pustules are frequently formed, first on the hands and then (probably by transfer of pus and serum by the patient's fingers) on various other parts of the body. Small acuminate papules are also very characteristic, and not less so are the scratch marks, often accompanied, especially in children, by wheals like those of urticaria. In severe cases of scabies the dermatitis may be intense, both hands and arms swelling as if with phlegmonous erysipelas; or arms, hands, legs, and feet may be the seat of weeping, raw surfaces like those of eczema madidans; the lymph glands of the axillæ and the groin become swollen and painful, and the excessive itching is at last replaced by the smarting and tingling of acute dermatitis. More often, especially in children, the pustules resemble impetigo or ecthyma, and form as they dry up thick scabs and crusts. In chronic cases—for unhappily we often see scabies which has lasted for weeks and months without detection and has been therefore ineffectually treated—the skin becomes thickened, indurated, hard, scaly, and fissured, resembling the condition of the more chronic forms of dry eczema. Bullæ as large as those of pemphigus are less frequent lesions of scabies, but are not uncommon in children. I have already referred to a case figured in the 'Guy's Hospital Reports' for 1877. In fact, we may say that all the inflammatory lesions of eczema, erythema, urticaria, pemphigus, impetigo, ecthyma, and lichen, may be more or less simulated. The large, flat, and discrete papules of prurigo, the imbricated scales of psoriasis, and the thin, dry, abundant squames of pityriasis ruba are never simulated by scabies.

Localisation.—The acarus itself infests the thin skin of the hands between the fingers, the flexure of the wrist, particularly its ulnar side, the flexor surface of the front of the forearm less frequently, the foot and ankle occasionally, the axilla and the groin, the genital organs, the inner part of the thigh, and the fold of the nates. But the lesions indirectly caused by its presence have a far more extensive though perfectly definite range; in fact the local distribution of scabies is so well marked that in a majority of cases a glance is sufficient to identify it. The inflammatory lesions are always present on the hands, except occasionally, when the patients are engaged in some handicraft which leads to the constant immersion of their hands in oily or strong-smelling substances or in metallic solutions, and forbids the development of the parasite. The same result is often seen in private patients, where the hands escape owing to the frequent use of soap and nail-brush. With these exceptions, which are important for diagnosis, the fingers and ulnar side of the wrist may be said to be the favourite seats of scabies as they are of the acarus. Some lesions will almost always be found upon the prepuce, and the inflammation usually affects not only the thin skin of the genitals, but that of the lower part of the abdomen, at least as high as the umbilicus. The whole of the forearms is very liable to be affected, and the eruption is more general than is the case with eczema. The axillæ seldom escape altogether, the buttocks are almost always more or less affected, and in children nearly constantly, especially near the thigh, but the perinæum and the sacral region usually escape. The toes, feet, and ankles, especially the inner ankle where the skin is thinnest, are very frequent seats of the dermatitis of scabies; less often the knees; the whole of the inner side of the leg and thigh may share in the inflammation. The back, shoulders, and chest are but little affected, the thick or hairy skin being apparently less favourable to the parasite. The neck, face, ears, and scalp almost invariably escape, in striking contrast to the frequency of eczema and impetigo in these parts. The only exceptions are in children, of which every experienced dermatologist must have noted occasional cases. Why the skin of the face is shunned is hard to say. It is not more exposed to the air than the hands, it is as thin and delicate and vascular as that of the abdomen, but for some cause it is shunned by the acarus. May it be that the large sebaceous sacs and thick cutis with thin cuticle which are characteristic of the face, as of the shoulders, the chest, and the scalp, furnish a fatty secretion which repels the invader?

It has often been remarked that a line drawn across the waist and elbows of a man standing in "the first position" will have below it the regions of scabies acarorum, and above it those of prurigo pedicularis.

In children, localisation of scabies is much less strict than in adults, as we found to be the case with eczema and psoriasis. In them only do we ever find the face affected; in them the hands frequently escape and runs are as well or better found round the ankle or in the skin of the sole; in them the trunk, and particularly the nates, are often more affected than the limbs.

Diagnosis.—This depends on a recognition, first, of the characters of the dermatitis, next of its very constant localisation, and thirdly of the cuniculi, the ova, or the acarus. The general facies of the disease is so characteristic that nine out of ten cases will be recognised in a moment when the patient is stripped, but in private practice anyone may be thrown off his guard who is accustomed to diagnose by probabilities rather than by facts. As I once heard Sir William Gull say, there are three diseases which we all sometimes overlook,—phthisis and syphilis and itch.

Where the inflammation has completely obscured all trace of acari the existence of the itch-mite may be proved by removing the crusts, boiling them in solution of potash or soda, and allowing the dissolved mixture to stand in a conical glass. On decanting and removing the deepest layer with a pipette fragments of the chitinous skeleton may be recognised.

It need not be said that scabies is always *contagious*, and its occurrence in an entire household often leads to its recognition. It is remarkable, however, how cases may remain isolated, and we must remember that impetigo and prurigo, not to mention variola and varicella, may also be contagious. The mode of transference is not always easy to follow; direct contact of hands is probably one method; often the ova are conveyed by clothes or other articles of constant use. Bedfellows seem particularly liable to infection. Frequently there is no doubt that scabies is strictly a venereal disease, the acarus having first invaded the genital organs.

Treatment.—Experience long ago discovered that sulphur is good for the itch; it appears to be an effectual poison to the acarus, and all we need is the best method of applying it.

The general practice is inunction of unguentum sulphuris into the affected parts, especially those which are the chief seat of the acarus. The colour of the application may be disguised, but its smell is always unpleasant. Sulphur lotions or sulphur fumigations may be substituted, but neither, I think, are so effectual. The best method is for the patient to rub the ointment well in every night, to lie in merino clothing all night with the ointment liberally applied, and next morning to wash with hot soap and water, and sparingly apply a little dilute sulphur ointment to the most irritable parts.

A rapid cure may be effected by first rubbing the skin with soft soap so as to remove crusts and epidermis, and then thorough sulphurous inunction. In this way patients are cured in a few hours at St Louis on a large scale, and their clothes meanwhile are baked and washed. This last precaution is important, since otherwise the patient may readily reinfect himself from his own clothing. With private patients the disease rarely gains such extension by neglect as to be severe, and its cure is usually quick and easy.

It may, however, happen that the sulphur ointment is itself too irritating; so that, although it kills the acarus, it perpetuates or sets up a fresh and even more severe dermatitis. One often sees these cured but overtreated cases of scabies, and all that is necessary is to recognise their nature.

With children, diluted ointment (two to one, or equal parts), in infants with much dermatitis, one to two are the best proportions, the dilution being made with benzoated lard or with zinc ointment.

In slight cases, especially in children, balsam of Peru is a pleasant and generally an efficient parasiticide.

ERYTHEMA

AND ITS ALLIES

Definition of the group—Its characters—The anatomical lesions—Course—Locality—Symptoms—Ætiology—Symptomatic and traumatic erythema—Pernio and pernio-like inflammations—Varieties (1) *Erythema multiforme—Herpes—Erythema iris*—(2) *Erythema nodosum*—(3) *Urticaria—Treatment of erythematous affections.*

RASHES PRODUCED BY DRUGS.—*The erythematous rash of Copaiba—Bromides and Iodides—Belladonna—Opium—Quinine—Salicylates—Arsenic—Mercury, &c.*

We have hitherto considered diseases which, though differing from one another in many particulars, are all examples of chronic superficial dermatitis; never leaving scars, chronic in course, apt to return, and accompanied with more or less decided irritation.

We now pass to diseases which form, I think, a natural group, though the line is perhaps more difficult to draw. They also are *superficial inflammations* of the skin and therefore leave no scars, but they are *acute or subacute* in their course. Moreover, their lesions are usually slight and evanescent, and although occasionally they produce one as conspicuous as a bleb, yet this is quite exceptional, and none of this group is attended with pustules or crusts. Perhaps the most conspicuous anatomical character is the presence of inflammatory *œdema.* The sensations accompanying them are usually *smarting* rather than itching.

Erythema is the name which has been given to some affections belonging to this group, and may be conveniently extended to the whole. The word, denoting "redness" of the skin, is used in classical Greek, either as *ἐρύθημα προσώπου* or alone, for a blush. It is also used in medicine almost, if not quite, synonomously with *ἐρυσίπελας.*

Willan classed erythema with roseola, urticaria, scarlatina, rubeola and purpura under the title "exanthemata," the general character of the order being hyperæmia of the skin without further lesion. Subsequent writers have called a mere hyperæmia "rose-rash" or roseola, while the word erythema, or, as Hebra calls it, *erythema exudativum*, has been confined to a rose-rash with palpable inflammatory exudation, diffuse or forming pimples. Hebra included also the so-called tubercular and nodose species of Willan's genus erythema, and invented a convenient term, *erythema multiforme.*

But in truth we have no need of a special title for mere hyperæmia, that is, dilatation of the blood-vessels without inflammatory exudation, such as follows division of a vaso-motor trunk in an animal. A mere blush is always a physiological phenomenon. Clinically, hyperæmia is, I believe, always inflammatory. Even the erythematous eruption of scarlatina, of measles, or of enteric fever can be proved by its course and sequelæ to be in each case true dermatitis. Bateman himself remarked that the efflorescence to which Willan appropriated the title of roseola is of little importance practically, and quotes the dictum of Fuller in his 'Exanthematologia' that it is "rather a ludicrous spectacle than an ill symptom."

We must recognise two meanings of the word "erythema" just as we are obliged to recognise two of the word "eczema." We saw that eczema is, as Hebra proved, a common superficial dermatitis, which has reached the stage of visible and usually serous exudation; but we saw also that the most important peculiarity of the disease eczema is that it is not traumatic, not called forth by ordinary irritants, and not limited by their action. An artificial or traumatic eczema is therefore for practical purposes better refused the name. Accordingly we added to the definition of eczema as a disease the character of being *idiopathic,* with its own peculiar distribution and course.

In the same way "erythema" may be defined, and has been used by Hebra and other authorities to denote the slightest form of dermatitis in which the classical signs of redness, heat, and pain are accompanied by little or no perceptible swelling. The irritation of a mustard plaister, for instance, will in most persons produce such a typical erythema, the scorching of the sun does the same, and if the skin be more than usually delicate and the mustard or sun more than usually strong, what was an " erythema " becomes an "eczema." It would be better, I think, if the term "superficial traumatic dermatitis" were used for both stages of the inflammation; or we might speak of the earlier as an erythematous and the latter as an eczematous or weeping dermatitis. Practically, however, little confusion is produced, even when the terms erythema and eczema are used in a double sense.

Just as we define the disease eczema by its clinical and pathological features apart from its mere anatomy, so we can define the group of affections which we have called erythematous. These are distinct from the erythematous stage of common dermatitis, and though often strictly "erythematous," sometimes exhibit other lesions, to which Hebra's adjective " multiform " very well applies.

I would therefore define erythema as follows:

1. The characteristic *anatomical lesion* is a rose-rash, resembling the first degree of traumatic dermatitis, that is to say *injection* of the surface, sometimes with obvious general *œdema,* sometimes with circumscribed œdema forming *wheals,* and sometimes with *papules* which are distinguished from those of eczema by not developing into vesicles, from those of lichen by their bright colour and transitory duration, from those of impetigo and prurigo by never becoming pustular, and from those of psoriasis by never becoming scaly. In some rare forms of erythema separate *bullæ* are formed which may simulate those of pemphigus. The rash is usually followed by a slight branny *desquamation.* It will be seen that, after all, the multiformity of the lesions of erythema is less than that of the lesions of eczema.

2. Whatever the nature of the lesion, *the exudation* is of a watery rather than a corpuscular nature, so that œdema, diffused or circumscribed, is its characteristic, in contradistinction to the sero-purulent or purulent vesicles and pustules of eczema and scabies. Moreover, along with the hyperæmia and œdema there is very apt to be a certain amount of escape of blood-corpuscles; an event which never occurs in eczema except as the result of direct injury, as by scratching. The result of this hæmorrhage is sometimes so marked as to give the title " purpura " to the eruption. Willan and Bateman rightly included *purpura urticans* with erythema among the exanthemata, although other kinds of purpura are altogether distinct from any form of dermatitis and are only parts of a general hæmorrhagic condition. The result of the hæmorrhage is to leave bruise-like pigmentation behind, so that this when present is very characteristic of true erythema.

In these characters, as in some others, and especially in the fact of the occasional occurrence of bullæ, the erythematous group bears a closer relation to pemphigus than to any other of the chronic forms of dermatitis belonging to the so-called dartrous group, but if one attempts to include pemphigus as an erythema its course and treatment forbid the conjunction.*

3. The *course* of erythema is subacute, that is to say it begins quickly, sometimes with slight febrile symptoms and does not last indefinitely. Even when its course is comparatively chronic, it will be found that the protracted disease is really made up of a series of outbreaks which may sometimes run into one another, but always preserve a recurrent or intermittent character. No erythematous disease ever acquires the chronic, stable, and inveterate stamp of eczema, lichen planus, psoriasis or pityriasis rubra.

4. The *locality* of the erythemata is much less definitely marked than that of psoriasis or of eczema. On the whole it is symmetrical, sometimes accurately and exclusively symmetrical, but there are frequent exceptions to the rule.

The favourite localities are, first, the extensor surface of the forearms and legs, especially the back of the hand, wrist, and ulnar side of the forearm, the dorsum of the foot and tibial side of the shin ;† secondly, the face, cheeks and neck; thirdly, the chest and abdomen. Lastly, the back of the trunk, the buttocks, thighs, and upper arms are least frequently affected; while the scalp, the flexures of the joints, the palms, and the soles are scarcely ever invaded by erythematous affections.

5. As a rule, smarting and tingling are the *symptoms* which accompany erythematous eruptions, while severe pain and itching are rare. Local tenderness is more marked than in eczema. Sometimes, and especially when wheals are present, the irritation is considerable, though never comparable to that of chronic eczema, scabies, or prurigo.

6. The erythematous rashes do not spread. They appear simultaneously at different spots, and fresh patches appear which may occasionally unite, but we never see the affected part of skin gradually enlarge its borders—the characteristic course of eczema and psoriasis.

7. Of the *ætiology* of erythema we in most cases know nothing. The lesion can, as above explained, be produced by moderate irritation, the diffused forms by heat or friction, those with wheals by a lash, or by the poison of the stinging-nettle, the hairs of certain caterpillars and the thread-cells of certain anthozoa. But in the non-traumatic, idiopathic, or "true" cases of erythema, the eruption can, in striking contrast to those of eczema and psoriasis, be in most cases traced to some *internal* disorder. In other words, erythema is usually *symptomatic*. The most striking instance of this is the erythematous rash produced by copaiba and by certain articles of food. Many other cases are dependent upon *dyspepsia*, others again upon *rheumatic fever*.‡ Moreover, one may fairly adduce in this connection the fact that the symptomatic early rash of syphilis, the exanthema of scarlatina and of measles, and the occasional prodromic roseola of smallpox, enteric fever, and cholera, all belong to the erythematous type.

8. Erythema occurs most commonly in children and young adults; it is comparatively rare after forty. Among persons past their prime it is less uncommon in women than in men. In these as in so many other points we

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* Since writing this chapter, I have observed that Dr Hans von Hebra, whose classification differs widely from that of his father, unites under the title Angioneurotic affections of the skin the erythematous rashes of infectious diseases, the rashes produced by drugs and poisons, and, thirdly, the essential erythemata, together with pemphigus and acne rosacea.

† Although the true homology of the tibia is undoubtedly with the radius and not with the ulna, yet the tibial aspect of the skin, from its having no underlying muscles, agrees pathologically with the skin covering the subcutaneous surface of the ulna, just as pathologically the patella answers to the olecranon, and the second metacarpo-phalangeal to the first metatarso-phalangeal joint.

‡ This is particularly true of E. nodosum, urticaria, and the hæmorrhagic forms of erythema known as *Peliosis rheumatica*.

observe a marked contrast to eczema and psoriasis, and a resemblance to pemphigus and also to rheumatism.

Before entering on the varieties and treatment of erythema proper, one must say a word of the traumatic dermatitis of slight degree which is still often called erythema. Thus *intertrigo* mentioned above under eczema (p. 620), is by Hebra and Neumann classed under erythema.

Common *chilblains* are a good example of chronic inflammation from the effect of cold and feeble capillary circulation of an erythematous anatomical type. The occasional bullæ which end in broken chilblains are not unlike those of erythema iris. Such chronic erythematous dermatitis may be seen not only in the fingers and toes of children and young persons during winter, but also on the ears and nose of a subject in whom, whether from central or peripheral causes, venous stagnation is apt to occur in the parts of the body most distant from the heart.

This kind of chronic venous congestion, which on the one hand may be quickened into chronic subacute dermatitis, and on the other may lead to hypertrophy, is seen in the blue and swollen ears, cheeks, and fingers of children with chronic bronchitis, and especially with bronchiectasis; in persons with chronic affections of the heart, especially of a congenital kind; in those patients, especially women, who habitually suffer from cold feet, and what they rightly call a languid circulation. It also is seen with somewhat different characters in the red, swollen, and irritable nose and cheeks which accompany dyspepsia, particularly, though not exclusively, in alcoholic dyspepsia, and is known as "acne rosacea" or gutta rosea (vide *infra*).

Lastly, a not dissimilar condition of cyanosis and chronic venous congestion with œdema is seen in those remarkable cases of symmetrical gangrene of the extremities which have from time to time been observed, and which are now spoken of as Raynaud's disease, since that physician called attention to the more severe cases which are connected with disease of the arteries, with embolism and sometimes with hæmaturia.*

The erythemata which are symptomatic of measles, scarlatina, enterica, rubeola, as well as choleraic roseola, and that which sometimes precedes the characteristic rash of smallpox,—all these have been described by Dr Fagge in the first volume of this work.

The erythematous and other rashes which follow the administration of drugs will for convenience be considered together at the end of this chapter.

There remain a group of skin affections which agree in the general characters of anatomy, course, and natural history described above, which are not traumatic, nor secondary to diseases of the vascular system, and which are not symptomatic, either of febrile diseases or of what the continental writers call "intoxication" with drugs or poisons. These we may style idiopathic, essential Erythema. Their common characters have been already sufficiently explained. It remains to point out three varieties which they present.

1. *Erythema multiforme.*—Simple or ordinary erythema, erythema papulatum, erythema exudativum.

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* Maurice Raynaud, 'De l'Asphyxie locale, et de la Gangrène symmétrique des extrémités,' 1862. A striking case of this remarkable condition occurred in Guy's Hospital several years ago under Dr Wilks' and my care. A boy about twelve years old had ulcerative endocarditis with emboli. This led to gangrene of the fingers and toes, and intermittent hæmaturia, with great pain and high temperature. He went out with the mutilated members healed, perfectly well except for a cardiac bruit. See also a case of Prof. Billroth's in the 'Wiener Med. Wochensft.,' 1878, reported in the 'Lond. Med. Record' of that year, p. 343.

The commonest kind of erythema is that which consists in general hyperæmia with œdema of the skin, a diffuse dermatitis which may either spread over a large surface with indefinite edges, or, as is more frequently and characteristically the case, occurs in patches with defined edge. On careful examination small papules may be often distinguished scarcely rising above the level of the skin; sometimes these are well marked enough to deserve the title erythema papulatum, but this is comparatively rare (a good example is the eruption of measles), and most lesions of the skin which receive this name are, I believe, either traumatic dermatitis or an early stage of papular eczema. Large, firm, and persistent papules such as occur in prurigo are never seen in true erythema. The inflamed patches have usually a very short duration; they may disappear in a few hours (*Erythema fugax* of Willan) and be succeeded by others, but if they persist for a day or two they may form rings which have been specially described as *Erythema annulatum* (*E. circinatum* of Willan), or *Roseola annulata.* When closely set several of these rings unite, and we have a sinuous reddish band produced which may be named *Erythema marginatum* or *E. gyratum.* Finally, the redness fades, the œdema subsides, and may leave no trace behind. If there is desquamation it is very slight and furfuraceous, more frequently a slight amount of pigment marks the seat of the eruption.

Erythema leve is a common dermatitis which is apt to appear upon the tense skin of dropsical parts, and may go on to deep dermatitis and sloughing. It is not uncommon as the result of acupuncture or of tapping, and is allied to traumatic erysipelas.

2. *Vesicular and bullous erythema.*—The exudation of erythema, instead of being a somewhat deep diffused œdema, sometimes appears in superficial collections of serum. These when small are called vesicular erythema or "herpes;" when large, "erythema bullosum."

Herpes, the common Latin term for an eruption of the trunk, in contradistinction to porrigo or an eruption of the head,* was limited by Willan to vesicular eruptions which he distinguished from the vesicles of smallpox and chicken-pox, from sudamina and from the inflammatory vesicles of eczema. His species of herpes included (1) *Herpes zoster* or zona, an eruption erythematous, it is true, in its anatomy and course, but which is so demonstrably connected with nervous disorder that it is rightly separated from all other forms of dermatitis, and was described in the first volume of the present work among nerve diseases (page 366). (2) *Herpes circinatus,* which we shall afterwards describe as the form which ringworm assumes when it affects the body, is a parasitic disorder, and is now classed with *Tineæ.*

Willan and Bateman's remaining species are *Herpes phlyctenodes* of uncertain seat, called *H. labialis* or *H. preputialis* when affecting the lips or the foreskin respectively, and *H. iris* when found on the back of the hands or the instep.†

These from their course and natural history, may, I think, be well included in the general group of erythemata This was, indeed, to some extent admitted by Hebra and even by Rayer before him.

Herpes labialis or *facialis* consists of a little group of vesicles upon a red patch of skin which appears almost suddenly, most often upon the upper lip. In a day or two the clear, pearly contents issue, become somewhat turbid and puriform, and dry up into a thin brownish crust which speedily falls off and leaves no trace behind. The vesicles entirely differ from those of eczema or ordinary traumatic dermatitis by their large size, by their not running one into another so as to form a weeping surface, by their acute course and by the sharply limited edge of the patch. They also are unattended with itching or pain and never consist of pure pus like the eruption of impetigo or scabies. Moreover, they are always symptomatic of some internal disorder, most characteristically perhaps of acute lobar pneumonia. Many persons are liable to such patches of herpes, either on the lips, or less frequently on other parts of the face, when they are attacked by acute catarrh. Bronchitis, catarrhal pneumonia, whooping-cough, asthma, are rarely accompanied by herpes. Sometimes the eruption appears to follow a rigor even when this symptom does not prove the precursor of pneumonia or catarrh.*

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* The word ἕρπης is derived from ἕρπειν. "Herpes dicitur eo quod videtur ἕρπειν, quod est serpere per summam cutem, modo hanc ejus partem modo proximam occupans." From the same creeping progress the disease was, according to Bateman, called *formica* by the Arabs.

† Herpes gestationis has been already described on p. 659.

This curious eruption has clearly little or no connection with eczema and its allies, nor can we link it with zona, for it frequently recurs, it is not unilateral, it does not follow the course of a nerve, and is unattended with pain. Its superficial character, sudden onset, and rapid course agree with the erythematous group as here defined, and the fact that it is symptomatic of internal disturbance and usually of irritation of a mucous tract completes the analogy.

Herpes preputialis when no longer left, as Willan placed it, and as Hebra was content to leave it, as a vesicular dermatitis, is difficult to classify. Hardy is even driven to the untenable assertion that it is nothing but local vesicular eczema. The rapidity of its onset and course, the superficial lesion, the patches, the absence of notable irritation or pain,† all point to its close connection with erythema, while its occurrence at the orifice of a mucous tract and the anatomical lesion bring it into still closer relationship with herpes labialis. The chief difference is that, occurring as it usually does on the inner side of the prepuce or glans the vesicles are broken almost as soon as they form and very superficial ulcers take the place of scabs. The condition is exactly like that of a vesicular eruption on the tongue. Like herpes labialis, it often recurs in the same patient; like it also it is often symptomatic of inflammation or stricture of the urethra, although it does not seem to be produced by cystitis and certainly does not follow inflammation of the kidney as labial herpes does inflammation of the lung.‡

Herpes iris is a rare and remarkable form of eruption well described by Willan, which is unmistakably erythematous in its nature and is in fact the type of a small but interesting group of cases which are best, I think, named erythema bullosum. It occurs sometimes as a single, sometimes as two or more rose-coloured patches with all the characters of erythema, almost always upon the back of the hand, the wrist, or extensor aspect of the forearm, more rarely on the corresponding part of the foot and ankle. It rapidly becomes annular, but before the ring is faded the patch of erythema reappears in the middle and may thus be surrounded with one or by a repetition of the process by two or even three concentric rings. This is the condition described by authors as erythema iris, but almost always the irritant patch becomes the seat of a large single vesicle or a group of smaller ones exactly like those of one of the patches of herpes labialis or herpes preputialis. The surrounding ring may exhibit similar vesicles or they may be more or less abortive, so that one might often question whether if we adopt the anatomical nomenclature we should describe the lesion as erythematous or bullous or vesicular. In its most striking form with a single large tense bleb like one of pemphigus, surrounded by vesicular circles, the whole patch as large as a crown-piece, it is one of the most remarkable of eruptions. The inflammation is very superficial, produces little pain or irritation, and after forming thin scabs passes off after a few days, leaving more or less pigmentation yet not a trace of scar behind. Partly the resulting pigment and partly the rosy red of the rings, the pearly grey of the vesicles and the more or less yellowish contents of the older bullæ seem to have combined with the bow-like form to give the title iris. The course of this curious disease, its superficial character, its locality, all make it unmistakable erythema, as also the fact that it occurs, I believe, exclusively in young persons, but so far as I am aware it is symptomatic of nothing.

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* See an interesting autobiographical account of a case of the kind by Mr Symonds in the 'Clinical Transactions' for 1884, p. 60.

† Pruritus may, however, be considerable at the beginning of the eruption.

‡ The maintainer of a thesis might, however, maintain that herpes labialis does not follow ordinary inflammation of the lung, and that pneumonia is not an inflammation of the lung at all, and produces herpes only as an acute pyrexia ushered in by a rigor. The chief practical importance of preputial herpes is its diagnosis from a soft chancre.

Iris is not, however, the only bullous form of erythema. Beside the form associated with imperfect circulation above described (p. 665) which is known as *pernio bullosa,* other cases of bullous erythema have been recorded by Dr Duffin ('Pathological Transactions' 1875), by Dr Crocker and Dr Frederick Taylor (Clinical Society's 'Transactions,' Feb. 25th, 1881), and I have reported two typical cases in the 'Guy's Hospital Reports' for 1880 (3rd series, vol. xxv, p. 211). But the best account of them is given by Mr Hutchinson in the 29th of his 'Clinical Lectures' on certain rare diseases of the skin: "of some peculiar eruptions allied to chilblains." Here also some cases of Bazin's Hydroa belong.

3. *Erythema nodosum.*—This curious affection was well described by Willan and Bateman, and subsequent authors have added little to their account. It occurs "in large oval patches, the long diameter of which is parallel with the tibia, slowly rise into hard and painful protuberances and as regularly soften and subside in the course of nine or ten days, the red colour turning bluish on the eighth or ninth day as if the leg had been bruised." In this form of erythema the anatomical lesion is especially characterised by œdema; the spots do not itch but are somewhat painful and very tender, more so than in any other of the erythematous group. There is almost always not only deep venous congestion of the typical erythematous rose tint exaggerated by its position on the legs, but there is almost always a slight indication of actual hæmorrhage. Probably from this cause the pigmentation, which like other forms of erythema it is apt to leave behind, is much deepened by the chemical transformations of the effused hæmoglobin which are familiar in a bruise.

The locality of erythema nodosum is, as Willan says, most frequently over the tibia, but it is not confined to this part, for I have seen it on the ankle and calf and it is not uncommon over the corresponding surface of the ulna. It is usually symmetrical and often affects the whole extensor surface of both forearms and both legs. It is very rare in any other part. It has a slower course than most kinds of erythema, but like them is prone to recur. Willan and Bateman and also Green in his 'Practical Compendium' state that erythema nodosum only affects women, but Plumbe in 1824 ('Practical Treatise on Diseases of the Skin') notes its occurrence in children, and it is not unfrequently seen in men, but almost always in those subject to hysteria chorea, or other female disorders, that is in boys under or about the age of puberty. It occurs very frequently in those who have suffered from rheumatic fever (see Dr Thos. Barlow, 'Brit. Med. Journ.,' Sept. 15th, 1883, p. 511). Dr Cæsar Boeck has also published a monograph on this point.

The course, the lesion, the œdema, the hæmorrhage, the locality, the subjects are all typically erythematous.

4. *Urticaria.*—Willan rightly classed urticaria in close relation to erythema. Almost all subsequent writers have followed this indication, and if convenience did not forbid innovations it might be called "erythema pomphosum," for the characteristic lesions are wheals, *pomphi*, raised flat white patches sometimes surrounded by an erythematous blush. Their histology is that of acute inflammatory œdema of the cutis which fills the lymph spaces and expels blood from the venules. The exudation takes place very rapidly and may be called forth either by a mechanical or by a chemical irritant as in the wheals produced by the nettle (*Urtica urens*) from which the disease receives its name. In persons liable to the affection it can be produced by the finger only drawn across the skin, so that it is possible to write letters in raised wheals. This last has been defined as "factitious" urticaria. The anatomical lesion has, therefore, its counterpart in those traumatic wheals produced in any skin by the sting of the nettle or the stroke of a whip, and produced with very slight irritation in susceptible subjects. The relation of such traumatic urticaria with the idiopathic disease precisely answers to that which I expounded at some length between common superficial dermatitis from the sun or other irritant and idiopathic eczema, between prurigo senilis a pediculis and idiopathic prurigo of Hebra, between erythema congestivum et bullosum and chilblains.

Beside the well-marked oval or linear wheals of ordinary urticaria we often see the lesion in the form of small round patches or in large white plateaux formed by the coalescence of a large number. Both these forms are frequently produced by nettles. I would also include as essentially of the same nature the large, flat, white papules which are obviously distinct from those of ordinary eczema, and which have been described as strophulus albidus, and also not unfrequently under the name of infantile prurigo. These papules are distinguished by rising rapidly, and by following, and not causing, pruritus, since they are the result and not the occasion of the patient's scratching. They are most often seen in infants, but may often be observed along with more obvious wheals in ordinary cases of urticaria.

The *distribution* of nettle-rash is less definite than that of other forms of erythema, and indeed of most other cutaneous affections. I cannot observe any predilection for the erythematous regions, the extensor surface of the forearms and legs. It is quite as common on the back and trunk generally as on the limbs; the only parts it avoids are the scalp, face, arms, and soles. It is not symmetrical.

Of all forms of erythema, urticaria is the most irritable, the severity of the itching being comparable to that of eczema, scabies, or prurigo. There is no pain or smarting, and no subjective symptoms except from the restlessness and sleeplessness which it occasions, especially in children. It is most frequent in them or in young adults, but is not confined to any age.

The *ætiology* of urticaria is uncertain. As above explained it is often purely secondary to some local irritant, and is probably always aggravated by the patient's scratching. Its close alliance with erythema is shown not only by sometimes alternating with it, but also by its following precisely the same kind of gastric disturbance, both in the most marked forms from drugs and poisons, and in the less evident cases of ordinary dyspepsia. Like erythema again it is, according to general experience, frequently connected with rheumatic fever.

Urticaria pigmentosa.—A singular form of skin disease belonging to the erythematous type, but as chronic as erythema nodosum, was first described by Mr Morrant Baker in the Clinical Society's 'Transactions,' vols. viii and x, and also by the late Dr Tilbury Fox, by Dr Sangster, Dr Cavafy, Dr Barlow, and myself. Two cases, one kindly sent me by Dr Goodhart in a child of two years old, and the second in a patient of Dr Fagge, of Ascot, will be found shortly described in the twenty-fifth volume of the 'Guy's Hospital Reports,' 3rd series, pp. 212, 213.

It has received several names, among others the uncouth and misleading term Xanthelasmoidea, but at present the best is probably Dr Sangster's proposed title, Urticaria pigmentosa.

It is an erythematous eruption with occasional wheals and considerable yellowish pigmentation lasting for an indefinite period, though I believe its chronic course is always made up more or less distinctly of subacute attacks. It affects the back and trunk generally rather than the limbs, is attended with pruritus, and has possibly some relation to rheumatic fever.

Treatment of erythematous affections.—The whole group of the erythemata show their true relationship from a practical point of view. They are none of them contagious, they are none of them attended with serious consequences, they are mostly indicative of some primary disorder, and they are rather to be palliated by local applications or indirectly cured by treating their internal cause when discovered, than met by a specific plan of treatment. In particular it may be said that they are either unaffected or aggravated by arsenic, and this is one of the points which separate them from pemphigus. In many kinds of erythema, especially symptomatic herpes and iris, no treatment is needful.

The local treatment of the erythemata consists in the astringent and sedative applications described at page 646; although the surface is dry, it is found by experience that lotions in most cases answer better than ointments. Goulard's wash, evaporating lotions, spirit and water, eau de Cologne, hydrocyanic acid well diluted, solution of borax are the best local applications. Warm baths should be avoided, as also excessive heat and perspiration; tepid water is better than either cold or hot. The patient should be urged to stoical abstinence from scratching; tepid bathing or firm, steady pressure will be found to relieve the intolerable irritation of urticaria without aggravating it afterwards as scratching always does. For the painful swellings of erythema nodosum, strong lead lotion gives most relief, or lead and opium. Collodion painted over and allowed to dry is often useful, or alum, tannic acid, or other astringent remedies may be used with advantage, or the affected part of the leg may be painted with a strong solution of nitrate of silver. A little zinc or zinc and lead ointment may be applied to the herpetic and bullous forms of erythema.

Internally, our first care should be to relieve the gastric disorder which often accompanies common congestive or papular erythema most often by discovering certain articles of food to which it is due. Salt fish, pickles, preserved fruit in the form of jams and crystallized sweetmeats, pork, sour or otherwise inferior wine, malt liquor, stone fruit, and even strawberries, any one of these may in certain persons excite erythema or urticaria. Those first on the list should be almost always forbidden. But of all kinds of food, lobsters and crabs, and by a curious coincidence mussels and other mollusks united with them under the title of "shell-fish" are the most frequent, the most certain and the most severe in their effects. Many drugs have a similar result, copaiba being probably the most effectual.

If the eruption continues after its supposed cause is removed, or if we are unable to discover any cause of disorder, such remedies as bicarbonate of soda with gentian or calumba or chiretta or a few drops of liquor potassæ in peppermint or cinnamon water should be prescribed. Where there is evidence of gastritis, bismuth is a most valuable remedy, given either in powder or thus: bism. subnitr., sodæ bicarb., pulv. tragac. co. āā gr. x., aq. chloroform. sive menthæ pip. ℥j. M.; to which ten or twelve drops of solution of morphia may be added if the pain is severe. With flatulent disorder, thymol, creosote, or carbolic acid in the form of pills are often the most effectual mode of treatment. In the more atonic forms, pepsin given before meals is found practically useful, notwithstanding our physiological doubts; and occasionally dilute mineral acids with nux vomica or bitter infusion will be more valuable than anything else. Gentle saline laxatives taken before breakfast in a large draught of warm water are almost always indicated; and, for women especially, a pill containing aloes or rhubarb taken before a late dinner or on going to bed is a useful adjunct. In many patients occasional doses of blue pill are of unmistakable value.

In cases of erythema nodosum and in other forms of erythema which follow rheumatic fever and occur in pale young women or lads, the preparations of steel are strongly indicated. When there is constipation, a good formula is three or four grains of sulphate of iron, half a drachm of sulphate of magnesia, and five drops of dilute sulphuric acid, in peppermint water or calumba. When this is not the case the tincture of steel is a most valuable remedy. In some patients sulphate of iron with carbonate of potash and extract of Barbadoes aloes forms the most valuable Martial remedy. In whichever form iron is found to agree best, it is important to increase the doses until a decided effect is obtained.

Eruptions produced by drugs.—Since the most frequent and characteristic effects of drugs upon the skin are erythematous eruptions, it will be convenient to consider this group of dermatoses here. Drugs and poisons act much in the same way upon the skin as do irritant or poisonous articles of food.

The most striking and frequent of these eruptions is perhaps that produced by *copaiba*, which has sometimes been confounded with an early syphilide. It usually takes the form of a papular erythema, often combined with urticaria and not unfrequently more or less hæmorrhagic. I have never seen bullæ or vesicles produced, but they are mentioned by trustworthy observers. In some cases there is no itching, which makes the diagnosis from syphilis the more difficult. The rash is generally distributed over the whole surface of the body and does not spare even the face, as most other erythemata do. Occasionally it simulates purpura. Some writers have suggested that it is not the copaiba but the urethral inflammation for which copaiba is commonly given which produces the rash. There can, however, be no doubt of the existence of a true copaiba rash. So far as I know it only follows the oleo-resin and has not been observed in persons who are taking the valuable diuretic, mistura copaibæ resinæ.

Cubebs is generally said to produce a similar eruption, but some of the reported cases appear to have been due to accidental mixture with copaiba. So at least Dr Bulkeley believes.

Somewhat similar rashes have been observed in patients taking *chloral hydrate, turpentine, cannabis indica*[*] and some other drugs; so that purpura, urticaria, pemphigus, herpes and erythema *a medicamentis* have been associated with these and many other drugs. I confess to scepticism as to iron being one of them, and some at least of the eruptions ascribed to salicylic acid were probably peliosis rheumatica.

Bromide of potassium comes perhaps next in frequency to copaiba as a rash-producing drug. The lesion here simulates very closely that which will be described in the next chapter as acne, but the diagnosis is generally clear from its not being confined to the very characteristic localities of true acne or to the equally characteristic age which is liable to that disease.

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[*] In a case reported by Dr J. N. Hyde in the 'New York Medical Record' for May 11th, 1878.

Less frequent but much more varied, more severe, and more misleading is the eruption produced by *iodide of potassium.* This is perhaps most frequently a papular erythema, widely or irregularly distributed on the trunk, limbs, and face without itching and usually unfelt by the patient. Sometimes, however, there is considerable erythematous dermatitis between the papules. A follicular inflammation undistinguishable from that described above as bromide acne is a less frequent effect of the iodide salts. More often the rash which was at first papular becomes vesicular, bullous, or pustular. In these cases the inflammation is often very severe and the constitutional disturbance considerable. They have been, there is no question, often confounded with herpes and so-called "idroa" or "hydroa" and indeed until one has seen several cases, it is difficult to believe that so severe a dermatitis can be due to a drug which in most cases has no effect whatever upon the skin. The eruption may simulate scabies or eczema, but the absence of definite localisation, of chronicity, of the secretion of eczema or of the cuniculi of scabies should make the diagnosis not difficult. Along with the pustules there may arise what the older dermatologists would have called a tubercular disease of the skin, raised fleshy nodules simulating papillary growths, condylomata, mucous patches, and the later forms of syphilodermia. I have seen them resemble rupia or lupus or even malignant disease. Inasmuch as these severe effects are apt to follow the large doses of iodide of potassium given in the later stages of syphilis the difficulty of discriminating them is naturally increased.

In a healthy man under my own care suffering from a very ordinary pustular syphilide, some of the lesions on the face and the back of the hand become so swollen, hypertrophied, and covered with profuse granulations, that both cheeks were deformed, the eyes almost occluded, and the back of one hand was covered with an exuberant granulated surface which when seen alone suggested to different observers lupus hypertrophicus or epithelioma. There was, however, no doubt of the nature of the case. My diagnosis was confirmed by several dermatologists and he recovered perfectly when the drug was discontinued. The chief point which guided me aright in this case was that, notwithstanding his frightful appearance, the patient was eating and sleeping well, so that it was with great difficulty I could persuade him to come into the hospital.

The rash is punctiform and resembles scarlatina rather than measles, the patches and rose tint of which are more nearly simulated by the copaiba rash. The iodide eruption is often purpuric (Dr Duffy, 'Dublin Journal of Medical Science,' vol. lxix, April, 1880).

Bromide and iodide pustules often appear and when deep and as usual occupying a hair-sac cause crops of boils. The presence of iodine and bromine has been actually demonstrated in the pustules by Adamkiewicz and Guttmann.* For an account of the histology see a paper by Dr Thin ('Med.-Chir. Trans.,' vol. lxii, p. 189).

Weeping dermatitis, curious wart-like nodules, and other peculiar eruptions have been described as the result of bromide of potassium by Voisin and Veiel quoted by Behrend ('Berlin Clin. Wochenschrift,' vols. xvi and xxii, pp. 626 and 714, 1879). Two cases of severe iodide eruption were figured by the late Dr Tilbury Fox ('Clinical Society's Transactions,' vol. xi, November 23rd, 1877).

The iodide produces its effects on the skin much more rapidly than the bromide, which is rare, except in cases of epilepsy, when it is used for months or years. In both cases there appears to be a true excretion of the drug through the sebaceous glands.

Various measures have been adopted to prevent these unpleasant effects.

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* 'Virchow's Archiv,' 1878, vol. 74; 'Charité Annalen,' vol. iii, p. 381, 1878.

The addition of carbonate of potash or aromatic spirits of ammonia is sometimes sufficient. Moderate doses of arsenic have been often recommended, but I have certainly seen them fail more than once in preventing the eruption. Changing the potash to the soda salt of iodine I have sometimes seen followed by the disappearance of the rash, but I doubt whether this was more than a coincidence. It is at least certain that persevering with the drug which had caused the eruption even in larger doses is often followed by the disappearance of the unpleasant effect.

Belladonna produces a bright red and almost universal erythematous rash. It may be recognised by its association with dilated pupils and a dry throat, together with the characteristic delirium when the dose has been large. I need scarcely remind the reader that children bear as large doses of this drug as adults, and are not more liable to these symptoms of intoxication. In one case under Mr Hilton I remember it was caused by the mere application of a large belladonna plaister in a woman who must, one supposes, have been more than commonly susceptible.

Similar rashes have been observed as the result of hyoscyamus or stramonium.

Opium and *morphia* sometimes produce considerable pruritus, but so far as I know any eruption which occurs is consecutive to the scratching which results. Opium rashes have, however, been seen by trustworthy observers. According to Behrend it also causes urticaria, but this I would suggest may be also secondary to pruritus.

Quinine.—There can be doubt that quinine may produce a general acute erythema, which was first described by Skinner, Fleming, and other English authors, and has been since observed abroad. Its symptoms closely resemble scarlatina. It begins in the face, spreads rapidly over the whole trunk, and is accompanied by severe fever, the temperature sometimes reaching 39·8° C. It is certainly a very rare effect of so popular a medicine, and seems to be an instance of true idiosyncrasy. In one case of Köbner's the eruption followed the exhibition of quinine three times in the same patient. A still more severe local erythematous rash of the face, sometimes vesicular, has been observed as the result of quinine by Hebra, von Heusinger, and some other physicians.

Morrow, who has collected sixty cases of quinine eruptions, found that in thirty-eight the rash was erythematous, in twelve it resembled urticaria, in two it was vesicular, and in five hæmorrhagic ('New York Medical Journal,' March, 1880). One case was reported by Dr Fagge ('Medical Times,' February 29th, 1868).

Eruptions from *salicylic acid** have been reported. Since erythema and urticaria are common in the disease for which salicylic acid is usually given, and since adulterations with carbolic acid and consequent gastric disturbances are not unknown, the interpretation of these cases may admit of doubt. The internal use of carbolic acid itself, of tar, turpentine, and petroleum have all produced rashes usually erythematous, but the cases are comparatively rare. An excellent bibliography of the whole subject is given in the text, and at the end of a paper by Dr Van Harlingen in the 'Archives of Dermatology,' Philadelphia, October, 1880.

Arsenic is said in some persons to produce an acute vesicular eruption which has been styled herpes. I have never seen this occurrence, although I am in the habit of giving rather large doses of the drug not only in cutaneous cases, but also in osteo-arthritis, chorea, idiopathic anæmia, and Hodgkin's disease. The fact that zona† has sometimes appeared during a course of arsenic, must, I think, be considered a mere coincidence.

If when a patient is taking arsenic and zona breaks out the fact that he has not been exposed to cold, and that the eruption is not epidemic may be accepted as evidence that arsenic is its cause, all inquiries into ætiology become at once easy and useless.

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* Cavafy, 'Clinical Society's Transactions,' vol. x, 1877, p. 88.

† Hans von Hebra, 'Die Krankhaften Veränderungen der Haut,' p. 204. He, however,

Mercury was one of the first drugs to be regarded as a cause of the cutaneous rash. Early in the present century Alley* described what he called Hydrargyria, before the first description of a copaiba nettle-rash by Montègre in 1814. Alley's cases were vesicular and corresponded with what we should now call eczema, chiefly of the abdomen, thighs, and scrotum, or sometimes of more severe pustular form, and still more rarely of bullæ with severe pain and lymphatic inflammation, sometimes combined with angina. It must be doubtful whether local inunction of the drug or the effects of syphilis itself, or a mere coincident attack of eczema may not explain these cases. Hebra, with a scepticism justified by his enormous experience, denies that any eruption on the skin is ever brought about by the internal use of mercury. The cases reported by Behrend, Lycel, and Engelmann were erythematous, and sometimes complicated with scarlatina. The question must still, I think, be regarded as undecided.

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is convinced that the relation is merely accidental. See also Hutchinson, 'London Hospital Reports,' vol. v.

* 'Observations on Hydrargyria,' Dublin, 1804; London, 1810.

DISORDERS OF THE SEBACEOUS GLANDS, THE HAIR-SACS, AND THE SWEAT-GLANDS

ACNE.—*Nomenclature—Anatomy and course of local lesions—Distribution—Age and sex—Symptoms—Ætiology—Treatment—Acneiform eruptions produced by tar—by bromide—Acne variformis—Acne tarsi.*

Milium.—Seborrhœa oleosa—Comedones without inflammation (acne cornée)—Seborrhœa sicca—Xerodermia—Steatoma—Meliceris and sebaceous cysts.

MOLLUSCUM CONTAGIOSUM.—*Name and history—Anatomy—Pathology—Treatment.*

SYCOSIS.—*Name—Anatomy and course—Locality—Diagnosis—Sycosis capilittii frambœsiformis—Treatment.*

FURUNCULI.—*Pathology—Anatomy—Course and distribution—Age—Contagion—Treatment—Carbuncle.*

AFFECTIONS OF THE SWEAT-GLANDS—*Anidrosis—Hyperidrosis—Bromidrosis or fœtid sweat—Chromidrosis—Hæmatidrosis—Sudamina.*

Still keeping to the long series of inflammatory diseases of the skin, we find certain affections which may fairly be arranged, on clinical as well as anatomical grounds, in a third large group. The first we considered was that of the chronic forms of dermatitis which in various degrees resemble the common superficial inflammation produced by irritants. Traumatic eczema, idiopathic, symmetrical, weeping eczema, papular eczema, lichen, lichen planus, pityriasis rubra and psoriasis—these form a natural group of which pemphigus is an outlying member. Although I see no reason for supposing that their pathological relationship depends upon the presence of an antecedent dartrous or arthritic or gouty diathesis, yet they undoubtedly are really related to each other. The erythematous group of affections treated in the preceding chapter, to which pemphigus may be considered as the link, form to my mind quite as natural though not quite so extensive a family.

The present chapter deals with inflammatory processes which do not affect the skin generally, but only the hair-sacs and cutaneous glands. They have also, as we shall see, peculiarities of distribution and of natural history which are no less characteristic than their anatomy.

ACNE.—This disease, of which the name is supposed to be a corruption of *ἀκμή* and to refer to its occurrence in the prime of life, was called *ἴονθος* by the Greeks, *varus* by the Latins, and is referred to by Cicero,* Martial, and other classical writers† as a blemish rather than a disease.

The sebaceous glands become occluded either by their secretion being too thick or by want of cleanliness in removing the accidental obstructions from dirt. The first effect is to produce a number of small, firm, and somewhat pointed papules (*comedones*), each of which is produced by accumulated sebum and is marked by a black head, which is nothing but the dirt obstructing the orifice of the gland. This condition, which has been named *acne punctata,* may continue for an indefinite time, but sooner or later some of the papules show signs of irritation, and in most cases this very speedily supervenes in each obstructed gland. The papule becomes red, swollen, and before long yellow from suppuration having taken place. This pustular form or pustular stage of acne is no less characteristic. When the surrounding inflammatory œdema is considerable the deformity is of course increased. At last the minute abscess bursts and the inflammation slowly subsides. When slight, no trace remains, but a second inflammatory process with the same course and termination often follows. When severe, a minute white scar is left behind, the gland is destroyed and incapable of renewed action. When the hair-sac into which the sebaceous gland opens is deep, the inflammation is the more severe, and sometimes causes a minute slough which leads to the pain and swelling characteristic of a furunculus. Such little boils are naturally slower in their course and lead to deeper scars. The face or shoulders may be seen covered with acne spots in all the above stages, so that the pain and irritation become great and the deformity distressing.

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* "Miro quid sit quod Servilius, pater tuus, homo constantissimus, te nobis tam varium reliquerit." Cic. fragm. ap. Quint. vi, 3, 48. *Vărus* is, of course, unconnected with *vārus*, knock-kneed, and may likely enough be connected with the adjective varius.

† "Pæne ineptiæ sunt curare varos et lenticulas et ephelidas; sed eripi tamen feminis cura cultus sui non potest." (Celsus, 'De Med.,' lib. vi, cap. v.)

Histology.—A section of an acne pustule shows not only the papillæ but the deeper layer of the cutis œdematous and filled with leucocytes, and the small blood-vessels dilated. In the pustular stage the leucocytes increase in number and assume the character of pus-corpuscles; the acini and duct of the gland are filled with pus, often mingled with blood-discs. The process in the larger acne pustules is found to affect the hair-sac into which the sebaceous gland opens, so that the hair itself is uprooted and the entire follicle destroyed. When the destruction of the papillæ has taken place—in other words, when the inflammation has become "deep" instead of superficial—a scar always results after the acne is cured.

Dr Liveing has found that in a sebaceous sac which is the seat of a comedo many minute abortive hairs may often be found, the growth of which may perhaps be the immediate cause of obstruction.

It should be observed that the presence of the minute parasitic mite known as *Demodex folliculorum* is frequent in healthy sebaceous glands and never causes acne.

Distribution.—Acne is confined, almost without exception, to the face, shoulders, and chest. It usually begins about the cheeks and forehead, the alæ of the nose and the chin; but pimples may cover the whole of the face and the intervening skin be occupied by an erythematous dermatitis. Comedones, as the black-tipped early lesions of acne are called, are also to be generally seen on the auricle, but here they rarely spread. On the back the pustular and indurated form is more common, perhaps because it is more apt to be neglected; and there it is that we see the most extensive cicatrices. The lesion may extend from the back of the neck and the scapular and interscapular regions down to the loins, but it seldom or never invades the sacral or gluteal region, nor does it pass round the flanks towards the chest and abdomen. Only a few scattered papules may be sometimes found over the deltoid or on the upper arm. The skin over the sternum is the least frequently affected of the three regions. The lesions are precisely the same and never extend to the abdomen, the axillæ, or the front of the neck.

Occasionally isolated comedones or acne-pustules may be found elsewhere, most often on the outside of the thigh and peroneal surface of the leg in burly men with coarse hairy skins and large follicles. These, however, do not occur particularly in the subjects of acne, and are either purely accidental or connected with inflamed lichen pilaris.

Age and sex.—This singular follicular inflammation is practically confined to the age of puberty for its origin and greatest extension, although acne when thus begun may continue up to thirty or even later. Comedones may be seen in a few rare cases (of which I have observed two) in children; but though numerous and apparently characteristic, they do not suppurate, and they are found upon the forehead and even in the scalp without the characteristic distribution of true acne. Again, a chronic inflammation of the skin, eczema, recurrent erythema, and especially that form which will be described below as gutta rosea, may lead to pustular inflammation of the hair-sacs, so that the latter affection has been commonly described as acne rosacea; but the distribution, the origin, and the whole natural history of the two diseases seem to me decidedly to separate them. In fact, the more closely the subject is studied the more decisively does true acne separate itself from all other affections.

The disease most commonly begins in lads of about sixteen, that is to say, when the changes which accompany puberty have already begun. It is not common for it to make the first appearance after the beard has begun to grow, but it may begin at from sixteen or seventeen up to one or two and twenty. It is very slow in its progress and the worst cases are usually those of a year's standing or more. When once thoroughly established, the morbid process continues even after the beard has fully grown, but in most cases it then begins to subside and seldom continues after the age of thirty. I have noticed that when acne occurs in a patient above this age, it is usually confined to the back and has been preceded by ordinary acne of the face. This is probably sufficiently explained, as well as the occasional occurrence of severe acne of the shoulders with only slight affection of the face, by the greater attention given to a visible eruption and the less efficient treatment of all affections which cannot easily be reached.

Although the evolution of acne is, as we have seen, so closely connected with that of the beard at puberty, yet the disease is very far from being confined to the male sex. Indeed, Erasmus Wilson stated in the first edition of his treatise that acne occurs more frequently perhaps in the female than in the male. This is certainly not the case, but acne is common enough in young women about the time or perhaps a little later than in lads. The affection is in them, I think, more diffused, the papules more numerous, not so large and with more erythema between. It is also more often confined to the face; and it is certainly much more rare to see the worst forms of acne indurata, and the disfigurement which follows, in women than in men. On the other hand, while it is somewhat later in its appearance it is decidedly slower in its disappearance, so that acne may be more often seen about thirty in women than in men, and it is chiefly in women that a lingering acne is overtaken by an early gutta rosea, a combination which has no doubt helped in confusing the two disorders.

Course and symptoms.—Acne is always a chronic affection lasting, if left to itself, for years, but liable to occasional exacerbations. These often coincide with ovarian disturbance in women; in men they are less marked, but sometimes appear to be connected with gastric disturbance, with indigestion, especially the more acute forms, such as in some people result from eating pork or salmon or preserved viands, whether salt, such as herrings, or sweet, like crystallised fruits and jams. There is but little local irritation, the other organs are completely unaffected; and indeed, but for the disfigurement, few patients with acne would apply to the physician.

Ætiology.—The immediate cause of acne in the obstruction and inflammation of the sebaceous glands, and in the severer cases of the hair-sacs also is proved, but when we ask why this obstruction and inflammation occurs, the answer is extremely difficult. To say that the presence of acne indicates a disordered state of the cutaneous nerves, which interferes with the vascular action of the skin; to say that it depends on torpidity of the capillary circulation or general want of cutaneous activity; to say with Biett that it is the result of keeping the head bowed down, as in many sedentary occupations, or the result of drinking cold water when heated, or of smoking tobacco; or with Alibert, that it is the result of spending nights in gambling and living in anxiety—all this is trifling with pathology. It may be asserted that acne has no such connection with feeble circulation, as is shown in chilblains, nor with local irritation as eczema solare, nor with gout or tubercle, nor with the ingestion of cold water or hot water, or alcohol, or any kind of food, nor with any diathesis or disposition to anything but acne.

It is obvious, if we consider its natural history, that acne has to do with the great change which passes over the organism at the time of puberty: first and principally with the growth of the beard, yet not as a mere mechanical result, for in the great majority of men the beard appears without acne,—men have acne who never develop a beard· and women frequently have it also. Acne, moreover, affects the skin of the shoulders, which is unchanged at this period, as well as that of the face and chest, where hair grows, and it does not affect the hair of the pubes. It is stated by Rigler ('Die Turkei und deren Bewohner,' Wien., 1862), quoted by Hebra, that acne, though common in the Levant, is extremely rare in eunuchs. Moreover, in young women affected with acne the eruption is often aggravated during the menstrual period. There is no reason to adopt the suggestion of Rayer, followed by many French writers, that acne is connected with vicious habits.

The old adage of Plenck, "Matrimonium varos curat" is well exchanged for Hebra's dictum, "Tempus varos curat." It is not continence nor vice, nor celibacy nor marriage, nor even the growth of a beard, which are the causes of acne; it depends upon the general changes which occur in the passage from childhood to adult life, the glandular apparatus of the skin is apt to be disordered, most apt on the region where the beard is developing and losing this aptitude when complete development is once attained. With regard to acne in women we can only say (as conversely of hysteria in men) that though they have no beards their fathers had, that is that secondary sexual characters are more or less transmissible to both sexes.

The question still remains whether there is any common condition of the skin in persons affected with acne. I confess I am unable to find it. It appears in those in good health and those in ill-health, in the blonde and so-called lymphatic, as well as in the dark atrabilious; it is said to be rare in persons with red hair, and to be less common in Ireland than in England.*

There is no proof that acne is a hereditary disease, although it is not unfrequently seen in brothers and sisters, and although the disposition to its development at puberty would, we might expect, be transmitted more or less completely in the same way as the early growth of a beard, its weakness or abundance, and the early or late supervention of alopecia or calvities. Dr Erasmus Darwin, who properly distinguished acne from gutta rosea (in his 'Zoonomia') nevertheless named the former affection *gutta rosea hereditaria*, "because it seems to be hereditary, or at least has no apparent cause."

Prognosis.—Few cases of acne cannot be decidedly relieved by careful treatment, and in many the face can be restored to its natural appearance; but success depends not only upon the physician's adapting his treatment to the wants of each case, but also upon the perseverance with which the patient will follow it out. In many cases irremediable mischief has already been done when the patient comes before us. "Tempus varos curat," though generally true, proves often tedious in performance, and when such a cure is complete the disfigurement it leaves is often considerable.

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* According to Bazin acne is of scrofulous origin, and even Hardy, whose admirable good sense always keeps him from the extravagance of his colleague, while denying this, thinks that acne has a preference for lymphatic subjects, although "on peut voir un tempérament lymphatique sans être atteint de scrofule."

Treatment.—In the early stage of acne, when comedones are present with little or no inflammation, the principle of treatment is to set free the obstructed ducts, to keep them clear by extreme care and to stimulate the local circulation. The plan I have found most successful is the following: On going to bed the face should be first steamed over a basin of boiling water. It should then be thoroughly washed with a piece of flannel and yellow soap, and dried with a rough towel. On careful scrutiny in the glass the patient will then find that the acne punctata has lost a good many of the black points; but he should go over the whole of the face, and wherever a pimple shows by the slightest point of yellow that suppuration has begun, it should be emptied—not by squeezing with the fingers, but by pressing over it the end of a key of suitable dimensions. When this has been effectually done the face should be again washed and a lotion used which should be allowed to dry. This drying lotion may be of sulphur suspended in liquor calcis, alum water or lead lotion, or a dilute solution of corrosive sublimate (gr. ij with tr. benz. co. ʒss in ℥j of mist. amygd.). The old cosmetic known as *lac virginum* was of somewhat the same composition, as was probably the famous Gowland's lotion, which is said by Bateman to have contained oxymuriate of mercury in an emulsion of bitter almonds.* The sulphur is the more stimulant; the mercurial wash when too strong is apt to cause a feeling of constriction and tension of the skin. Next morning any fresh pimples which have ripened should be emptied and the face again washed with soap and water and a little dilute mercurial ointment (ung. hydr. ox. rubri with two parts of benzoated lard) applied to each. With many patients thorough washing and the application of white precipitate ointment serves the same purpose very well.

The same plan of treatment answers, even if a good many pustules are present, supposing that there is not much inflammation around them; but the more pustules there are, the less vigorous should be the friction used and the more important it is to apply some ointment containing mercury to the pustules. Ung. hydrarg. amm. or citrine ointment are often well borne; in other cases the unguentum metallorum suits better.

When the inflammation, judged of by the erythema between the papules, by the amount of swelling, or by the presence of true furunculi, is severe, we must begin with other measures. Steaming is still useful and generally proves soothing, but friction must be much more sparingly used, and sometimes omitted altogether. Instead of a stimulating lotion or ointment the patient must at bedtime use Goulard wash or a drying lotion of oxide of zinc suspended in water, or the almond wash may be used alone; during the day lead ointment or zinc or a combination of the two must be applied, but for women and others who are not obliged to be out the frequent application of lead lotion is better than ointment.

It is in these cases only that diet needs regulation by abstinence from stimulants, spices, and the other viands which we found tend to excite erythema of the face (page 672). It may also be desirable for the patient to take a little carbonate of soda or citrate of potash with a saline laxative By these means the erythematous inflammation will soon be subdued, and it is desirable as quickly as may be to return to the more stimulant treatment.

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* "Merely Gowland," said Sir Walter Elliot, "I should recommend Gowland, the constant use of Gowland, during the spring months." ('Persuasion,' vol. ii, chap. 4.)

In inveterate cases the stronger mercurial ointments are indicated. When, as occasionally happens, large furunculi are present, they must be treated with carbolic oil, and if necessary with poultices.

Acne of the shoulders, though often severe, is naturally less troublesome than that of the face; the skin is also less susceptible to irritation and almost always bears rougher treatment with advantage. The individual attention to the several papules which is so important in the case of the face is of course much more difficult to carry out here, and we must depend more upon the use of mercurial ointments and on friction with rough towels or flesh gloves.

Acne tarsi.—Anatomically allied to acne is the inflammation which not unfrequently, especially in children, affects the large and specially modified sebaceous glands that serve to lubricate the eyelashes. These Meibomian glands are apt to become the seat of chronic inflammation, when a gummy secretion is exuded which sticks the eyelids together. It may either occur independently or as a complication of catarrhal or other forms of ophthalmia. It is generally cured by the application of unguentum hydrarg. ammon. or yellow oxide of mercury ointment.

Tar and bromide acne.—Inflammation of the sebaceous glands, papular or pustular, is occasionally called forth by external irritants, and especially by tar. This so-called tar-acne differs, however, in its distribution and natural history from the true disease, and needs no treatment but the removal of the exciting cause.

Bromide acne is the name given to a somewhat similar follicular inflammation caused by the internal use of the bromides in certain patients. It has been treated of above (p. 678).

Acne varioliformis.—This name was given by Bazin, who has been followed by other French authors, to what will presently be described as *molluscum contagiosum.* It is unfortunate that Hardy has accepted such a confusion of nomenclature and of pathology. The name has, however, been since applied to a rare and curious affection,* of which I have seen three or four cases. It consists in large pustules resembling those of the more severe kinds of acne, and situated chiefly upon the forehead, the temples, and the sides of the cheek. After they have burst and healed a deep scar is left, sometimes pitted but not pigmented, resembling that which follows the most severe forms of acne. It is in these scars that the resemblance of the affection to variola chiefly consists, for the distribution, the course, the absence of a vesicular stage, and the unimpaired health of the patient, could never allow of its confusion with variola. In a patient of my own, a man about forty, the affection encroached upon the scalp, and also spread to a considerable part of the chest, shoulders, and back. In this, as in the other cases which I have seen, this curious affection was quite distinct from true acne. It is not preceded by comedones, and it seems doubtful whether the pustules are really seated in the sebaceous glands. The distribution and the severity of the eruption also distinguish it from acne, and it is unconnected with the period of puberty. It is more difficult to distinguish it from a pustular syphilide, and some cases which have been described as acne varioliformis are, I believe, really syphilitic. In one case of my own, in which there was neither history nor proof of venereal disease, the pustules, which had lasted for a long time, disappeared under iodide of potassium. We must wait for further observations before the true nature of these cases can be decided.

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* Neumann, following Hebra, calls it acne frontalis, and describes it as a variety of true acne. Dr Bulkeley, of New York, names it acne atrophica or lupoid acne. See a case figured by Dr S. Mackenzie in the 'Clinical Transactions' for 1884, p. 227.

It will be convenient to refer briefly in this place to less important affections of the sebaceous glands than acne.

Comedones are generally the first stage of that disease, and only occur in the persons and under the circumstances above described; but beside the accidental comedo or even pustule which may be produced here and there by accidental obstruction of a duct on any part of the body, and which no more make acne than one swallow makes a summer, there are occasionally to be seen large numbers of comedones in children, affecting the forehead, scalp, and other parts, not undergoing inflammation and without the locality or other characters distinctive of acne.

Acne cornée is the name given by French writers to this remarkable and rare condition, to which I should refuse the name of acne, first, because it is not inflammatory, and, secondly, because it has not the natural history of the disease of that name. It consists in the presence of a multitude of *comedones*, which remain as passive papules, hard pointed and black tipped. They occur in children before the age of acne and upon the scalp and other parts unaffected by acne. I have seen several cases of this singular condition, which requires to be distinguished from "lichen pilaris." Once it occurred on the forehead and scalp of two brothers, once on the temples of a boy of eight or nine suffering from pleurisy, and once on the lumbar region of a girl, aged thirteen, under my care for erythema multiforme, but without a trace of acne or even comedones on the face, chest, or back (see 'Guy's Hospital Reports,' 3rd series, vol. xiii, p. 213).

Milium.—A commoner condition is passive obstruction of a sebaceous with complete occlusion of the orifice. A minute white or yellowish papule is thus formed without the pointed top or the black mark of a comedo. It has been called milium. It never inflames and is of no practical importance; it occurs most often on the thin skin of the eyelids and the genitals; occasionally it grows larger than a pin's head; when this occurs it usually affects only a single milium. Its contents then not unfrequently become liquid and it forms a small cyst such as may be occasionally seen on the eyelids and have been noticed by Mr Hutchinson to occur in association with xanthelasma and with sick headaches.

On dissection the acini of the gland are found filled with a dark refracting substance, which yields on analysis cholesterin, olein, palmatin, and stearin. A good drawing is given by Neumann, Fig. 10.

Seborrhœa.—The functional disorders of the sebaceous glands may lead to too abundant liquid secretion, or to too solid scaly products, or by suppression to abnormal dryness and harshness of the surface. The first of these conditions has been named seborrhœa or steatorrhœa. It is physiological at a certain period of life, when it forms the *vernix casiosa* of newborn infants. It is not uncommon about the face and especially the alæ of the nose. It also occurs on the genitals, where a local vernix casiosa may lead to pruritus and inflammation: cleanliness and a little lead lotion or ointment is sufficient treatment.

Seborrhœa sicca appears to depend upon the more solid fats, stearin and palmitin, being secreted in greater abundance than the liquid olein. The secretion forms little yellowish scales, added to by the natural desquamation, and frequently by the local irritation of a slight dermatitis which increases the desquamation. The condition is most common on the scalp, where it constitutes what is known as pityriasis capilitii, dandriff, or scurf. In most cases this is rightly termed seborrhœa sicca; but, although it begins as a sebaceous affection, in cases which have lasted long, one finds that the scales consist in large part of epidermic cells, and there is often beside local irritation, injection and other signs of dermatitis. That this is secondary is shown by its not spreading beyond the scalp and by its being unconnected with eczema or psoriasis of the scalp. Beside the irritation of this common disorder it undoubtedly leads to the hair becoming thin and weak and in some cases produces early alopecia.

The treatment is not very satisfactory. Mild mercurial ointments or carbolic oil sometimes prove useful.

Xerodermia.—Diminution or absence of sebaceous secretion leads to the skin being dry, harsh, and apt to crack. The sweat-glands may be active and abundant; but sweat is the more apt to exert an irritant effect upon the skin undefended by its natural oily secretion. This condition is usually congenital and was rightly described under the name "xerodermia" by Wilson as the first and slightest stage of ichthyosis. It will be again referred to under that head.

A similar state of skin is, however, not unfrequently observed in children who are thin and ill nourished and in patients of any age suffering from prolonged wasting diseases, especially phthisis. The diminution of subcutaneous fat is accompanied with diminished supply of oily material to the sebaceous glands and the skin becomes dry, pale, rough, scaly, and dirty. This condition, which is of only symptomatic interest, has been described as *asteatosis* and as *pityriasis tabescentium.*

The only treatment indicated is to supply the deficient oily material by inunction with olive- or cod-liver oil.

STEATOMA.—When the orifice of a sebaceous gland is obstructed and an accumulation of the secretion takes place, it does not always inflame; the secretion may go on until a large cystic tumour is formed. The orifice of the duct can still usually be found, and sometimes by mere pressure the contents can still be evacuated. They consist of inspissated sebum without, on the one hand, the pus which mingles with the secretion in inflamed acne, and without, on the other hand, the remarkable modified epithelial cells which will presently be described as characteristic of molluscum. When the watery parts are absorbed, the sebaceous secretion consists of the ordinary animal fats, palmatin, stearin, and olein, some butyric and caproic acids, either free or united with glycerin to form neutral fats, a small amount of albumen or rather globulin, with a larger proportion of casein, epidermic cells, with the flat tabular crystals of cholesterin, and earthy salts. Occasionally the fatty material appears to be absorbed as well as the water, and there remain behind only calcareous masses like those found in a diseased aorta or in the apex of the lung in a case of obsolete phthisis. These cutaneous calculi are, however, of very rare occurrence. The yellow, somewhat granular, half liquid, and half solid mass has been compared with putty and with mortar. It is exactly what the Greeks meant by atheroma, and although this term is now generally applied to the very similar products of chronic inflammation of the arteries, yet atheromatous tumour is still used by some writers as synonymous with what is otherwise called *Steatoma melliceris* or, perhaps better, a sebaceous cyst.

These tumours, called, when they attain a large size, *wens*, most frequently occur upon the scalp, where they are often multiple and may grow to the size of a fist or even bigger. They may also be seen upon the eyebrows, face, and neck, less frequently on the trunk, and most seldom of all upon the limbs.

Cysts with similar contents but of very different origin and, probably, (some of them certainly) congenital, occur on mucous membranes and in deeper parts of the body, especially about the root of the tongue and hyoid bone: and such cholesteatomata have also been described in the brain and in the bones. Many of them are true dermoid cysts and may be compared with those of the ovary, which contain not only sebaceous matter but hairs and sebaceous glands.

Sebaceous cysts are perfectly innocent, but occasionally require removal from their inconvenience or unsightliness. The plan usually adopted is to incise the tumour and tear or dissect out the secreting cyst wall, or, if this be difficult, to rub the interior with caustic.

Molluscum Contagiosum.—This somewhat rare disease was first described by Bateman, who added it to the small group of tubercula defined by Willan. The case figured by him in his 61st plate, on the face and neck of a young woman, was a typical example of the disease, and Bateman traced the contagion from a nursling of this patient and two other children in the same family to a fourth patient as the origin. He also mentions a second case in an infant apparently received from an older child. It was from these facts that the epithet *contagiosum* has been applied by English physicians,* and also by von Bärensprung, Virchow, and Rindfleisch, and although the correctness of the epithet has often been doubted it is now satisfactorily proved.

Carswell, Rayer, and other writers recognised Bateman's disease, and Huguier, in 1846, had described it as a non-syphilitic affection of the vulva. Caillaux, in his 'Treatise on Diseases of the Skin in Children,' named it acne molluscum.

The disease occurs in the form of small rounded tumours of a pink colour, sometimes sessile but more often pedunculated. They are scattered irregularly over the skin, which remains quite healthy between them. Their number varies from a single tumour to a countless multitude, and the size from that of a vetch, to use Bateman's comparison, or a large pin's head, to that of a marble or occasionally much larger dimensions. The colour also, though usually pink and waxy, is sometimes scarcely distinguishable from that of the skin, and at others it has a dead white or even yellowish tint. These last, however, are probably not uncomplicated examples of the disease. A minute dimple is to be found on each tumour, which points to the orifice of a sebaceous duct. This disputed point, however, will be presently considered. The growth of the little tumours is very slow. They may retain a size not exceeding a pea while increasing in number during many months, and perhaps longer. As they grow bigger they become more rounded and the groove at their base becomes deeper until they may be united by only a slender pedicle, a condition which was formerly described as acro-chordon. The colour usually becomes paler and more translucent as they increase in size, but this is not constant.

Distribution.—They are most common on the face and neck, especially upon the eyelids and cheeks, and also upon the mamma of women, but where a multitude of small molluscous tumours occur I have found them upon the arms, especially the thin skin of the flexor surface, as well as on the face.

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* Wilson, 'Diseases of the Skin,' 1842, p. 302; Paterson, 'Edinburgh Medical Journal,' vol. lvi, 1841, pp. 213, 280. Cases were also recorded by Alibert, Biett, Cazenave, Schedel, Gibert, and Jacobovitz, 'Le Molluscum: Recherches Critiques,' Paris, 1840. Most of the foreign cases, however, were examples not of Bateman's disease, but of what will afterwards be described as molluscum fibrosum.

The term molluscum, there is no doubt, was, as Mr Wilson suggested, taken by Bateman from the celebrated case of Tilesius described by C. F. Ludwig, of Leipzic, in 1739. His words are, "Corpus tactum est verrucis mollibus sive molluscis.' The word is obviously used as a synonym of mollis just as mollusca was first applied to the mollusca nuda et testacea, the *soft*-bodied animals or malacozoa. Alibert, followed by Cazenave, misinterpreted the meaning of the term. Bazin unfortunately described the disease under the term *Acne varioliformis,* and this has led to much confusion, especially since the same term has been employed for a singular variety of acne mentioned above (p. 684) before its misapplication to molluscum contagiosum had been forgotten.

No symptoms are produced in the most marked and typical cases; as before stated, the skin between the little tumours is perfectly normal, and they themselves are no more than a disfigurement.

They are most common in infants and young children, but I have seen them in adult males. They also occur not infrequently in women on the breast as well as the face, but molluscum of the mamma has, I believe, been never observed except in women during suckling.

Molluscum has been described as also occurring on the male genitals, but in the cases I have seen the tubercles have been yellow and not either pearly and translucent nor pink and waxy looking, and on incision or even on pressure without incision have yielded opaque yellow oily material, so that they should undoubtedly be called steatomata. In the Guy's Hospital Museum, Model 496 shows the appearance of molluscum upon the thigh. Sometimes a large molluscous tumour will suppurate, burst, and thus cure itself. A case of this kind, which occurred under the late Dr Addison, in a little girl ten years old, was modelled by Mr Towne for the same museum.

Anatomy.—On incising the tumour, a white opaque thick material can be usually squeezed out, and a hollow sac remains flaccid behind. Herein a molluscum tumour resembles an ordinary sebaceous cyst or steatoma, but the contents are white instead of yellow, and to the naked eye have not the atheromatous appearance so characteristic of accumulated sebum. Moreover, tested chemically and microscopically, instead of fat, cholesterin and earthy salts and epithelial scales, the white material seems to be made up almost entirely of characteristic oval transparent bodies with a pearly lustre without a nucleus and not readily staining with logwood. These have been described as molluscum corpuscles. They were first recognised and well described by Wilson in 1842; they were rediscovered at St Louis and described as cryptogamic spores, the source of the contagion. This view, however, is undoubtedly incorrect. Their size, their aspect, their reaction to potash and their inability to develop are conclusive against it. I have no doubt that they are epidermic cells which have undergone a hyaline transformation. When, as is sometimes the case, along with these molluscum corpuscles there is found a certain amount of fatty sebaceous material, we must, I think, regard the condition as one complicated by the addition of the sebaceous secretion. However, I have certainly seen in some well-marked cases an entire absence of sebaceous matter.

A section horizontal to the surface shows that each little tumour is made up of loculi more or less separated from each other by septa, and in most cases a central axis may be demonstrated, which is supposed to be the duct of the gland. The question remains whether the origin of the cyst is in the sebaceous gland, and whether the metamorphosed epithelial cells are derived from those lining its acini or from those of the duct, or whether the whole tumour is a new growth unconnected with the sebaceous apparatus and starting in the deeper epidermic cells. The latter is the opinion held by Virchow and supported by some excellent observations of Dr Sangster. I confess, however, that I am not convinced on the point; but must content myself with referring to papers by Drs Morrison, Crocker, and Thin in the 'Pathological Transactions' for 1881, p. 245, and also to a description by Dr Davies-Colley, 'Guy's Hospital Reports,' 3rd series, vol. xviii, 1870, p. 350 and figs. 1 and 2, p. 364. He describes most of the characteristic oval cells as nucleated. Virchow's original paper was published in 1865 in the 33rd volume of his 'Archiv,' p. 144.

Mr Hutchinson describes undoubted molluscum as not uncommon on the penis and scrotum of young adults, and in one case mentioned by Dr Paterson similar tumours were apparently caused by contagion upon the vulva of a woman whose husband was thus affected. He also refers to the molluscous growth occasionally suppurating, and points out that it occasionally may resemble an indurated cancer.

Molluscum contagiosum is most common in infants and children, less so in women, and decidedly rare in men. In children it almost always affects the face, in women the mammæ, and in men the genitals. But this is only true of the larger and fewer tumours. All the cases of numerous very small molluscum simulating warts and sometimes described as molluscum verrucosum which I have seen in adults have been situated on the arms. Mr Hutchinson has observed similar cases in which the trunk or lower extremities have been so covered with little tumours as to resemble some papular eruption as lichen. It appears to be much more common in England than abroad, though cases of the true disease are reported from France and Germany. It is well known in Scotland and also in America. The majority of cases occur in dirty neglected children, but I have often seen it in those who were clean, rosy, plump, and in every respect healthy.

Ætiology.—There can, I think, be no doubt that the epithet contagious is rightly applied to this disorder, notwithstanding the frequent failure produced by inoculation and the incredulity of Hebra and other dermatologists, who have not so frequent opportunities of observation as occur among the out-patients in London and Edinburgh. The subject is well discussed by Dr Duckworth in two interesting papers in the 'St Barth. Hospital Reports' for 1868 and 1872. No fungi and no bacteria have yet been observed.

Treatment.—Molluscum tumours beside, as above stated, sometimes suppurating, which appears particularly apt to occur when they are confluent, may also undergo passive involution by gradually shrinking and subsiding. This must be the case with many infants who have never come under medical treatment, but these modes of spontaneous cure are rare or slow and uncertain, while treatment is rapid and efficient. Each tumour must be removed either by snipping off with a pair of sharp scissors curved on the flat, or by being incised and emptied. In each case the whole of the diseased structure must be removed. Where the tumours are very numerous, it may be better to apply nitric acid or the acid nitrate of mercury to each one in the early stage. When the growths are not larger than pins' heads Mr Hutchinson believes that white precipitate and sulphur ointment in equal parts will cure the affection, but it is rare for an opportunity for this treatment to occur.

SYCOSIS—*Syn. mentagra.*—Closely allied to acne is a disease which consists in pustular inflammation of the large hair-sacs of the beard. In fact, anatomically it is difficult to draw a broad line between them, for although we speak of acne as inflammation of the sebaceous glands, yet since all these glands open into a hair-sac, obstruction of their duct and obstruction of the corresponding hair-sac are almost the same. In fact, the acne pustule when fully formed may be as truly said to occupy the hair-sac as the sebaceous gland. The difference anatomically lies in this, that whereas on the general surface of the body the hairs are small with shallow sacs, and the sebaceous glands large, in a man's beard the hair-sacs are large and deep. Moreover, there is no doubt that in acne it is the gland which is first obstructed so as to form a comedo and which afterwards inflames, while in sycosis it is the hair-sac which is the primary seat of disturbance.

The name *sycosis* was applied to this affection by the Greeks, from its supposed likeness in the worst cases to the inside of a ripe fig, the red pulp answering to the inflamed and swollen skin, the seeds to the little pustules. "Est etiam ulcus quod a fici similitudine *σύκωσις* a Græcis nominatur."

(Celsus, lib. vi, cap. 3.) Another term, *mentagra*, still sometimes used, was applied by Pliny (lib. xxvi, cap. 1) to a disease of the skin which has been assumed to be identical with what we now call sycosis. Mentagra and lichen menti of Pliny and Martial, like the sycosis of the Greeks, were certainly often applied to syphilitic affections of the lips and other parts, and so to what we now call mucous patches and condylomata. The Latin term *ficus* was certainly applied not only to condyloma ani but also to hæmorrhoids. It was Bateman who first accurately defined the characters of sycosis in the modern sense of the word. He placed it under Willan's order Tubercula on account of the swelling and induration which often surround the pustules.

Anatomy.—Hebra has expressed the opinion, and it is supported by Liveing and other observers, that the immediate cause of a sycosis pustule is the presence of two or more hairs growing together in the same follicle. Wertheim, in 1861, published a paper in the 'Transactions' of the k. k. Ges. der Aerze of Vienna, in which he referred the origin of sycosis not to the growth of more than a single hair in the follicle, but to its being abnormally thick.

Hebra, after reviewing the statements of previous authors, affirms that in more than 300 cases of sycosis or "follicular inflammation of the beard" he has never seen a single case accompanied with parasitic fungi. There can, however, be no doubt of the existence of what the French writers call parasitic sycosis. I have seen many cases at St Louis and also, though much more rarely, in London. I never saw a case in Vienna, and I believe that those who have studied dermatology in several schools will agree with me that this is one of the points in which we must admit the same local differences as, for instance, led formerly to the controversy concerning typhus and enteric fever, which depended upon the former being predominant in Edinburgh and the latter in Paris. Parasitic sycosis certainly does occur and will be found if looked for in London, but it is far less common here than in Paris; and it must be added that the presence of the fungus is often only to be ascertained after prolonged and repeated search. When detected, however, the case acquires at once a new character, and for practical as well as scientific reasons it is, I think, desirable to separate parasitic sycosis from the non-parasitic disease and it will be referred to as ringworm.

Whatever the immediate cause, suppurative inflammation takes place in the hair-sac, the hair-bulb becomes loosened, but the shaft still blocks the sac. The drop of pus first formed is pent up and produces pain and fresh inflammation. By the time that the hair is at last detached a small but deep cutaneous abscess has formed, and considerable congestion and œdema around it has produced what Willan called an inflammatory tumour or nodule. When the pus at last finds exit it dries into a scab which is rendered much more adherent than that of impetigo by the numerous hairs which tether it to the skin. Fresh accumulations of pus take place beneath it, and thus in severe cases a most repulsive and "malignant" aspect is produced by sycosis.

Course.—If left to itself sycosis is a most obstinate disease. The hair-sacs are successively destroyed and cicatrices result which are sometimes deep and obvious. When at last the disease has worn itself out the greater part of the beard is often permanently destroyed and the face disfigured by scars. The affected part is usually very tender, though, except when touched, there is rather tension and heat than severe local pain.

Excluding parasitic sycosis, which is of course contagious, Hebra and most German writers maintain that inflammation of the hair-sacs of the beard is non-contagious; but this, I think, cannot be affirmed. As before stated (p. 637), pus is itself in many cases an extremely contagious product. We see this upon the skin in cases of contagious impetigo and of furunculi, and I think it is wise to consider all cases of sycosis as more or less capable of spreading by inoculation of neighbouring hair-sacs in the same person or even under favourable conditions to another person.

Distribution.—As explained above, the peculiar character of inflammation here described can only occur in the large and deep hair-sacs of the beard. The disease usually begins upon the chin, frequently on the upper lip, on the cheeks, or under the jaw. It may, however, occasionally be observed in the eyebrows and on the pubis, and still more rarely what perhaps may be fairly called sycosis has been observed upon the chest, thighs, and other hairy parts.

Bateman asserts that women are not altogether exempt from sycosis, and Wilson admits that in rare instances it has been seen in the female. This, however, is, I think, a question of diagnosis, or at least of definition rather than of fact. The typical disease, excluding acne, inflamed ringworm, impetigo, and all syphilitic affections, is confined, if not absolutely, with the rarest exceptions, to the chin, lips, and cheeks of male adults. It is, I think, rare to see it in the comparatively soft beard which has never been shaved. However this may be, if the beard is suffered to grow without interference from the razor sycosis will not occur until it has become thick and coarse.

Parasitic sycosis.—Since Gruby, in 1847 ('Gazette Médicale,' No. 37), published an account of a cryptogamic plant which he discovered in cases of sycosis, French writers have generally described sycosis as a parasitic affection. Bazin named it Teigne Mentagre. Biette placed sycosis, reasonably enough, among pustular dermatoses, and was followed by Cazenave. Gruby's name for the fungus was *Microsporon mentagraphytes*, but Bazin and Rubet proved that the fungus is identical with trichophyton tonsurans, the name given to that of common ringworm in 1846 by the Swedish writer Malmsten. So also Kobner in 1864. Hardy follows Bazin and diverges from Cazenave in practically maintaining that all sycosis is parasitic, for he describes the teigne sycosique and has no place for sycosis among the dartres or elsewhere.

*Sycosis capilitii.**—This title of Willan's should perhaps be given to five remarkable cases of sycosis, or pustular eczema, of the hair-sacs observed by Hebra on the occiput and nape of the neck, and these may possibly be identical with Alibert's, unless we suppose with Bateman that these were mismanaged porrigo favosa, or with Hebra that they were undiagnosed syphilis.

These cases of Hebra's form the subject of a paper by Kaposi called "Dermatitis papillomatosa Capilitii." Hans von Hebra ('Archiv. für Derm. und Syph.,' 1869, p. 382) describes them, under the name suggested by his father, of *Sycosis frambœsiformis.* He describes it as "höchst selten," and as beginning in very small somewhat red papules, each traversed by a hair, which grow together and form growths resembling raspberries (*Himbeeren*) of extraordinary hardness, and at last ending in a long, tough, cheloid-looking band. The disease occurs on the nape of the neck where the hair is growing; the skin around is eczematous and red, and the place painful. The course is very slow, and as new papules arise they fill with thin pus. When raised flat papules have been formed hairs are seen pushing out in bundles. This, with the hardness of the growth, its extreme slowness of development, and its locality are the characteristic points. It may, however, occasionally occur on the scalp. This same affection has been described by Dr Alfred Vérité as *Acné kéloïdique* (Académie de Médecine dans la Séance de 9 Mai, 1882). And Mr Morrant Baker has described a case of it with an excellent drawing under the same title ('Pathological Transactions,' 1882). It is most common in the male sex.

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* The sycosis capilitii of Willan, p. 66 of his 'Atlas,' is not unlike the curious affection described on p. 684 as *Acne varioliformis.*

The treatment of this remarkable disease consists in destruction with caustics or removal of the tumours while still small with a sharp spoon, by galvano-caustic, or by other means. When the disease has already gone far excision is the only remedy.

Histological sections show that there is true enlargement of the papillæ, very scanty exudation of leucocytes, and gradual formation of parallel and interlacing bundles of fibrous tissue, among which the sebaceous and sweat-glands are squeezed and atrophied.

Diagnosis.—Sycosis must be distinguished from eczema of the face, ordinary papular eczema or impetigo, which sometimes invades the cheeks and lips, and simulates sycosis. It is, I believe, possible for the dermatitis thus produced to penetrate to the deep hair-sacs, and then a condition ensues which must be termed true secondary sycosis. But this is certainly very seldom the case. The superficial dermatitis preserves its superficial character, the pustules and crusts are those of impetigo, and when removed leave the surface but little affected. The treatment by ung. hydr. ammon. is simple and rapidly successful. No scars are left behind, no hairs are destroyed.

Treatment.—In cases of true sycosis which affects the hair-sacs, mercurial ointments must be combined with epilation. It is not, however, necessary in most cases to remove all the diseased hairs, and certainly not healthy ones. It is enough if those which are already loosened are extracted, so that the rule is for the patient to pluck out all the hairs which will come easily and without pain, that is, those which are already detached from their sacs. The first step in severe cases is to steam the face, if necessary, to soften the crusts with poulticing, sweet oil, and then to remove them with a broad pair of forceps. The beard should be cut short, but not shaved. If there is much local pain and swelling the inflammation should first be subdued by lead lotions, lead and zinc ointments, and other soothing and astringent applications. When this is accomplished the treatment above advised should be begun and followed out day by day. In most cases the result is successful. When cure has resulted it is generally better for the patient not to shave for several months, but to allow the beard to grow naturally.

Not unfrequently, however, sycosis proves very obstinate in spite of all care and diligence. The possible presence of a parasitic cause should in such cases be carefully looked for. In the most obstinate cases complete epilation on Plumbe's and Hebra's plan is no doubt the only effectual treatment, but it should be carried out piecemeal and with the help of previous application of potash-soap, and other remedies which soften and loosen the hair. During epilation dilute red oxide, or better, perhaps, the yellow oxide of mercury ointment should be rubbed into the surface.

Bateman recommended the unguentum hydrar. nitr. one in three, or white precipitate ointment with an equal part of zinc or lead.

Furunculi.—*Boils.*—Recognising the complete impossibility of any complete and satisfactory classification of skin affections which can set forth all their complicated mutual relations it seems to me convenient to associate the troublesome and painful affection of boils with acne and sycosis, for although it is not possible to demonstrate that the seat of inflammation is always in a hair-sac yet in many cases this may be readily ascertained and I think it probably true of all. However, the depth of the inflammation, its pustular character, and the scars which it leaves behind, are points in which it is closely related to the affections described in this chapter and particularly to the deep and painful suppuration which affects the vibrissæ of the nostrils and the ears.

The characteristic pathological feature of furunculus is that the inflammation leads to the death of a minute portion of the deeper layer of the cutis. This slough or core of necrosed connective tissue is passed out by a process of liquefaction and suppuration, and the abscess which is formed then slowly heals. In its early stage the disease appears as a pimple, distinguished by its excessive pain, a pain which resembles that felt from the plucking out of a hair, and in all probability depending upon inflammation of a hair-sac under somewhat different conditions from the comparatively painless pustules of acne and sycosis, The papule speedily shows a yellow spot in its pointed summit and this little pustule is never preceded by even a transient vesicle. Meantime, a bright, intensely injected halo appears around the pustule and considerable inflammatory œdema swells the whole skin into a conical elevation; the pain increases and becomes throbbing in character, while the dull constant aching and sense of tension is varied from time to time by sharp stabbing pains. When the abscess has ripened and is lanced or bursts of itself the core becomes visible, and is sometimes not expelled for a day or even longer. This stage is accompanied by a sharp pricking pain which is very characteristic; the pain rapidly subsides when the core is got rid of, a small scab forms, the redness and œdema disappear, and soon nothing but a minute scar remains. Unfortunately, however, it is seldom that this process is confined to a single furuncle. Most often a second and a third appear before the first is completely healed, or a whole crop may spring up almost simultaneously. A succession of painful abscesses may thus be established and last for weeks or even months until the patient's health seriously suffers from the pain and the discharge. Sleeplessness, loss of appetite, and much depression, both physical and mental, may be the result. When a crop of boils thus appears they are generally found to vary in size and severity, from those which are so large and deep as to challenge the name of carbuncle, to small superficial pustules which the older dermatologists would have called ecthyma.

Distribution.—There is scarcely any part of the surface which may not be the seat of a furunculus, but the affection has nevertheless a decided predilection for the back of the trunk from the hair at the nape of the neck to the fold of the nates. The thick cutis, thin epidermis, and small but numerous hairs of the dorsal region appear to furnish the most favourable conditions for this kind of inflammation. To this extent the distribution coincides with that of acne. The back of the neck, especially at the edge of the scalp, is perhaps the most frequent seat of all; the skin over the scapulæ is certainly less often affected; the buttocks come next to the nape of the neck. Moreover, the friction of the collar of the dress in the latter situation, and that occasioned by walking and sitting in the former, aggravate the misery of the complaint and probably keep it up.

Boils are far from unfrequent in the coarse skin of the outer part of the thighs which resembles that of the dorsal and scapular regions anatomically. They may also appear, though less frequently, on the leg below the knee, on the upper and forearm, on the wrist, where I have seen many cases, and on the back of the hand. The chest and abdomen, and even the face, are not exempt, but I have never seen boils on the scalp, the palms, or the soles of the feet. They are rare on the male genital organs, but not unfrequently occur in the neighbourhood of the anus, in the perinæum, and on the vulva.

Ætiology.—The true cause of this form of inflammation is unknown. There is, I believe, no ground for supposing that boils are the result of indigestion or of overwork or exhaustion from any cause; when numerous and long continued they produce, but are not the product of, anæmia and weakness. Nor, on the other hand do they come from plethora and over-richness any more than from poverty of the blood. In the practice of the water cure it is customary to wrap patients who suffer from dyspepsia, paralysis, and most other chronic diseases in wet sheets, which are then surrounded with blankets; free perspiration is thus produced, and in many cases it is offensive in odour and accompanied by a copious crop of boils and pustules. This is supposed to indicate the efficiency of the plan in bringing out the poison from the system, but in reality it only means stimulation of the sudoriparous glands, possibly vicarious excretion of urinary or fæcal products, and traumatic inflammation of the skin.

Age.—Boils are far the most frequent during youth, They are rare in infants and not common in early childhood, but schoolboys are very liable to them, especially to the most characteristic form of successive crops on the shoulders or on the nates. After thirty, liability to this painful affection becomes decidedly less and it is comparatively rare to find boils in an elderly man. Women during the whole of life seem less liable than men, though some of the most severe cases I have seen have been in young women.

I have no doubt that boils are sometimes contagious from one patient to another; and almost an epidemic of boils may sometimes run through a school, but more frequently from place to place upon the same patient.

Treatment.—If this view be correct it furnishes an important indication for treatment. While the first furuncle is developing, no doubt a poultice gives great relief from its warmth and the relaxation of tissues it produces, but by making the skin sodden and softening the epidermis it predisposes it to inflammation and renders the access of the chemical or morphological contagium of the pus more easy. I am persuaded that the constant application of poultices over large surfaces affected with boils very much tends to spread and continue the disease. A much better plan is to dispense as much as possible with poultices, using the water dressing instead, to apply to the skin immediately round the boil the lotio plumbi, Goulard wash, or a somewhat stronger lead lotion. Tannic acid may be used with the same object, or as each bulla appears a circle may be drawn around it with tincture of iodine or dilute solution of silver. Sometimes collodion, especially the flexible collodion applied in the same way, seems to act best both as an astringent and a protective. Meantime, the pustules should be covered with lint soaked in carbolic oil (one in ten) and the same antiseptic dressing should be continued after the pustule has burst. It was formerly the practice to open each boil successively with the lancet, but though the process is undoubtedly thus hastened, the pain and the dread of the pain are so severely felt, especially in young people, and when a sensitive part of the body is affected, that at least in such cases I think the furunculi may be left to ripen and burst of themselves.

I can see no special advantage in the purges and alteratives which are the traditional treatment for persons affected with boils. Where only one or two exist no internal treatment is necessary, but where the crops are numerous and successive the treatment above indicated by local astringents and antiseptics should be combined with the internal administration of wine or porter with the meals and of either bark with mineral acids or tincture of steel in full doses. Thin delicate boys will often be much benefited by cod-liver oil either alone or, if anæmia indicates it, in combination with steel. There is, reason to believe that a stay at the seaside is particularly useful during convalescence.

Carbuncle.—This term, as the diminutive of *carbo*, is the Latin translation of ἄνθραξ, a coal, and was applied to any red, angry, inflamed pustule.

The word anthrax has in recent times been applied to the very characteristic disease known as splenic fever accompanied with a characteristic boil or carbuncle of the skin, but always derived by contagion from cattle and always associated with the presence of a specific bacillus, which has been fully described in the first volume (p. 318). A carbuncle is pathologically identical with a boil, differing only in its severity and extent, but its natural history is sufficiently different to justify the old distinction being retained.

Anatomically a carbuncle is the inflammation which accompanies a considerable cutaneous and subcutaneous slough. It differs from the larger and deeper boils by the affected tissue being so extensive that not a single pustule and opening forms, but several, giving a characteristic perforated aspect to the broad summit of the tumour. If left to itself this gradually opens by ulceration and a deep and wide aperture is formed through which the slough is at length extruded, often with considerable hæmorrhage. The surrounding redness is commonly deeper and more lurid in hue than that of a boil; the œdema also is more extensive. A carbuncle almost always occurs single. Its most frequent seat is the nape of the neck and the shoulders; it may occur on any part of the trunk, but is rarely seen on the limbs or the buttocks. Occasionally it appears upon the face and is then severe and often dangerous.

Affections of the Sweat-glands.—The sudoriparous glands are less liable to disease than the sebaceous, and their affections are less important from a local, though far more important from a symptomatic point of view.

Anidrosis, or deficiency of sweat, is seen as a concomitant of many forms of pyrexia and usually accompanies erythematous and roseolous eruptions. In most of the forms of superficial dermatitis, also in psoriasis and in pityriasis rubra, little or no sweat is secreted, and probably the same is true of eczema madidans. The skin of ichthyosis, including its slighter forms which will afterwards be described as xeroderma, dry skin, are also marked by absence of sweat. That the function of this secretion is only supplementary to that of the kidneys and lungs as an excretion of water, and that its chief purpose is not excretory but regulative of temperature, is shown by the fact that patients with universal ichthyosis or pityriasis rubra show no symptoms of blood-poisoning from retained excreta.

Hyperidrosis.—General or profuse secretion of sweat takes place under two conditions. First, along with hyperæmia; this occurs in health during the natural sweating and warmth of skin induced by active exercise, and pathologically in rheumatism and the sweating stage of ague. Secondly, profuse cold perspirations take place with an anæmic state of the skin, as in the cold perspirations of terror, the night sweats of phthisis, and the cold perspiration which sometimes marks the approach of death. Modern physiology teaches that the vascular supply and the epithelial activity of sweat-glands as of other secreting organs are governed by two distinct sets of fibres, the former of which appear generally to belong to the ganglionic, the latter to the directly spinal system of nerves.

Local hyperidrosis particularly affecting the hands and feet is sometimes the source of considerable annoyance. Astringents are often useful, particularly tannin and alum. In one of the most troublesome uncomplicated cases I have seen profuse perspiration of the palms of the hands in a somewhat delicate young lady was cured, after other treatment had failed, by the local application of belladonna. Internally the same drug is indicated by our knowledge of the physiological action of atropine upon the submaxillary gland, but the difficulty is in pushing the doses sufficiently far without disturbing the general health, or rather in substituting for an occasional large dose the altered habit which may be produced by continuance of less than physiological doses for a considerable period.

Bromidrosis and *osmidrosis* are awkward names given to fœtid perspiration which is usually also excessive. This most frequently affects the feet and may then become a source of the utmost discomfort. The persons it affects are almost always young adults, and women more frequently than men. The horrible stench results from decomposition of the fatty matter which mingles with the sweat, and it is particularly the fatty acids which form the earlier terms in the series of which formic acid is the first—butyric, caproic, and caprыllic which give rise to it. Dr Thin has described and figured a bacterium to which this decomposition is probably due (' Proc. Royal Soc.,' 1880, No. 205).

The treatment of this distressing affection is often extremely difficult. The first step is to check the secretion by astringents and to prevent its soaking into the clothing by absorbent powders such as lycopodium ; the next is by frequent change of linen to remove the products of excretion as rapidly as possible. Antiseptics like thymol and salicylic acid may be usefully applied, and the latter preparation, especially in the form of collodion or a salicylic plaister, has the further advantage of softening the accumulation of macerated cuticle which helps to keep up the disease. With the same object Hebra used to recommend the careful envelopment of the foot and toes in strips of diachylon plaister, and I can testify to the efficiency of this plan of treatment, the details of which will be found minutely given in the New Sydenham Society's edition of his work, vol. i, p. 89. Dr Thin found a saturated solution of boracic acid the most efficient (' Brit. Med. Jour.,' Sept. 18th, 1880).

Almost the only other seat of evil-smelling sweat beside the feet are the axillæ.

Chromidrosis is the name given to the occasional secretion of coloured sweat. The sweat of the axillæ always contains in some persons enough pigment to stain their linen of a reddish tint. I have seen one well-marked case of this, and Hoffmann has recorded another in the 'Wiener med. Wochenschrift' for 1873, No. 13. Sometimes, however, a bluish pigment stains the sweat on the face or elsewhere. Cases of supposed chromidrosis occurring in young women should be watched. In most cases the apparently dark sweat is an *arte factum*. But although all the cases I have seen have proved factitious, there is no doubt that true chromidrosis does occasionally occur ; and in some cases it has been proved to depend on indican, a not unfrequent constituent of urine (p. 351), being excreted in the sweat and turning to indigo blue when oxidised by exposure to the air. Dr Foote published a case in the ' Dublin Quarterly Journal ' for August, 1869, and collected no less than thirty-seven others. Another source of colour is the production of blue or greenish fungi in decomposing sweat. When coloured sweat affects the eyebrows, it is usually of black colour, looking almost like soot. A remarkable case of red-coloured sweat was reported by Dr Wilks in the ' Guy's Hospital Reports ' for 1872. In this case a chemical analysis by Dr Thomas Stevenson proved the presence of iron but the absence of hæmoglobin.

Uridrosis, or the excretion of urea in the sweat, probably only occurs as a morbid phenomenon. The observations of Funcke on the normal excretion of urea through the skin have not been confirmed, but in Bright's disease it is not unfrequent to find even visible discharge of urea in the sweat (see p. 457 of this volume).

Hæmatidrosis (or *hæmidrosis*), a bloody sweat, is an extremely rare but undoubted morbid condition. It does not appear to accompany purpura or other diseases in which one would anticipate such hæmorrhage from general changes either in the blood or the capillaries, and in some of the very few authentic cases on record it appeared during apparent health, as in the hand of a friend of Hebra's, where he observed the exudation of blood-stained sweat upon the hand while sitting at table.

Dysidrosis is the name given by the late Dr Tilbury Fox* to a curious affection of the skin of the hands, since described as cheiro-pompholyx by Mr Hutchinson.† It consists in large vesicles without any surrounding inflammation, occurring in groups upon the palm and back of the hand and the fingers, especially near the web. These vesicles have been compared to sago grains, though they sometimes reach a much larger size. Rasori, who published a case in the 'Transactions' of the International Medical Congress for 1881, vol. iii, p. 146, calls it hydro-adenitis diffusa. Hans Hebra records a case under his care in August, 1882, in his 'Kr. Veränderungen der Haut,' p. 426. It affected the palms and soles of a woman forty-two years old, and some of the larger bullæ were surrounded with a red halo. Mr Hutchinson has observed relapses of this singular affection on several occasions. Whether it depends, as Dr Fox supposed, upon obstruction of the duct and accumulation of its contents, and if not, what its true pathology and relations are, remains a mystery. Dr Liveing regards it as an inflammatory affection characterised by the symmetry of the parts attacked, its most frequent limitation to the hands and feet, the vesicles or blebs, and its tendency to recur. He has also noticed that the nails are sometimes undermined and broken near the root. He notes a typical case observed in America by Dr Robinson, of New York. The few cases I have seen incline me to agree with Dr Liveing, that this chiro-pompholyx is rather allied to a bulbous erythema than dependent on obstruction of sudoriparous glands.

Sudamina.—There is, however, a well-marked though uncommon cutaneous affection which depends upon accumulation of sweat in little vesicles under the skin and has been known for centuries as sudamina or miliaria. It is only seen during the profuse sweating of rheumatism or other general diseases. The orifice of the duct becomes obstructed by some mechanism which is not very obvious, and the horny cuticle is raised as a thin transparent layer enclosing a drop of tranparent fluid, miliaria crystallina. This ruptures before it exceeds the size of a pin's head, but sometimes the contents become turbid and alkaline from slight inflammation. On the chest and back these sudamina are not common; they never occur on the face, and are rare on the thick skin of the palm, where they naturally attain larger dimensions before they burst, so as to resemble the "sago-grain" vesicle of chiro-pompholyx above described.

The profuse sweat which causes sudamina also produces, especially if not quickly removed, local irritation. This seldom goes beyond the stage of papules or erythematous redness except where it is aggravated by friction. The commonest seat for this *dermatitis a sudore* is the vertebral groove from between the shoulders to the sacrum, and the front of the chest. The more severe inflammation which occurs sometimes in the axillæ, often between the toes, and most frequently in the perinæum and between the cheeks of the nates, has been already described (p. 635) as intertrigo.

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* 'Skin Diseases,' p. 476.

† 'Lancet,' 1876, vol. i.

RINGWORM AND ITS ALLIES WITH OTHER AFFECTIONS OF THE HAIR

RINGWORM.—(1) *Tinea tonsurans—Anatomy—Course—Events—Histology—Detection of the fungus—Ætiology—Prognosis—Treatment—Parasiticides—Irritants—Mode of application—Epilation—Precautions against contagion*—(2) *Tinea cincinata—Form and locality—Burmese ringworm—Tinea marginata—Treatment of ringworm of the body*—(3) *Onychomycosis—Rarity and obstinacy.*

FAVUS.—*History—Anatomy—The fungus—Treatment.*

ALOPECIA.—(1) *Physiological*—(2) *Febrile and syphilitic*—(3) *Area—its appearance—locality—spread—question of its parasitic nature—prognosis—diagnosis*—(4) *Universal alopecia*—(5) *Congenital alopecia.*

TRICHOCLASIA *or brittleness of the hair.*

TINEA VERSICOLOR.—*Names—Parasitic nature—Appearance—Distribution—Diagnosis—Treatment.*

We have seen in other parts of this work that, beside animal parasites, the human body is liable to the invasion of the lower forms of vegetable life. The Schizomycetæ, often spoken of generally as *Bacteria,* are by far the most important of these, since they probably form and certainly convey the contagion of some and possibly of all specific fevers. They are described at p. 22 of the first volume.

Of far less practical importance are the fungi or mycetæ which are parasitic on the human body. Some of these affect the mucous membranes and have been already described in the present volume as *Oidium albicans* in the mouth, p. 99, and *sarcina* in the stomach, p. 153. We have now to consider diseases of the skin which depend upon the growth of similar microscopic fungi. In most cases the cryptogamic spores and mycelium lodge in the deep hair-sacs of the skin.

We will take, first, the most important of this group, then the remaining parasitic affections, and, lastly, it will be convenient to deal in this chapter with non-parasitic affections of the hair which need to be distinguished from ringworm.

RINGWORM OF THE SCALP.—*Tinea tonsurans,* the *Porrigo scutulata* of Willan.—This troublesome disease, which our forefathers described as "ringworm," and rightly distinguished from "scald head" or impetigo of the scalp, was only proved to depend on the presence of a cryptogamic parasite in 1844 by Malmsten, the Swedish microscopist. He named the fungus *Trichophyton tonsurans.*

Origin and spread.—We seldom see the earliest stage of the disease, but the first effect of the entrance and growth of the fungus in the hair-sacs is for the affected hairs to lose their glossiness and colour and become dry, shrunken, and brittle. They break short and probably thus expose fresh spores to spread the contagion. At the same time, the growth of mycelium in the hair-sac produces slight irritation, partly from the inflammation directly excited and partly from the patient's scratching. Moderate hyperæmia and corpuscular exudation follow, so that by the time a small bare patch appears, it is raised, slightly red and covered with a few scales. The process extends partly by the spores being conveyed to fresh places, partly by their steady advance to the next adjacent hair-sacs. Thus, one, two, and often numerous round patches are developed, each of which closely resembles the other. The form is often geometrically circular, sometimes oval or irregular; the hair is replaced by a few broken, dark, and thick stumps, which can be recognised by the naked eye, while their characters are still more obvious under a lens; the surface is usually covered with greyish yellow desquamation composed of epithelial cells and sebaceous material mixed with broken hairs, spores, and mycelium. It has a uniform, granular, closely adherent appearance which is almost decisive to a practised eye. At the edge of the circle a little redness may sometimes be observed, occasionally a few papules, and still more rarely, a vesicle or two. In the immediate neighbourhood individual hairs may be found by the aid of the microscope to be already affected by the spreading evil.*

A ringworm patch may increase to several inches in diameter without materially altering its appearance, but more often it is modified as it expands.

Either from scratching or from the effect of the fungus on the naturally irritable scalp, or as the result of irritant applications, more or less of ordinary superficial dermatitis appears, so that many cases of ringworm appear as impetigo capitis, and their true nature is not manifest until the scabs and crusts have been removed. In neglected cases, moreover, pediculi are not unlikely to breed and further aggravate and confuse the condition. Such horrible masses of felted hair, mingled with inflammatory products, vegetable and animal parasites, and all kinds of filth, constitute the *plica polonica* of Eastern Europe, which may still be sometimes seen at Vienna.

On the other hand, if the hair is kept short and the head clean, and if the skin is not naturally irritable, the fungus while spreading at the edges of the patch, appears to exhaust the soil in the centre and dies away like the larger cryptogamic fungi which form fairy rings upon the grass. The result is that the middle of the patch is more or less completely bald, with only a few short stumps or thin, feebly-growing hairs, while the circumference is occupied by a zone of flat brownish scales, more granular desquamation, papules and broken hairs. This is the most typical form of traditional ringworm and probably suggested the specific title *scutulata*. When growing patches of the disease meet, they form figures of 8 or dumb-bell-shaped patches, and as they still grow and unite with others, irregular gyrate figures like those of old-standing psoriasis, of erythema marginatum, and of syphilodermia. At last almost the whole scalp may be invaded and reduced to baldness. There is, however, never a perfectly smooth clear skin left as in alopecia areata, but a few ill-developed, thin, pale, scattered hairs are always to be found. Moreover the process is seldom or never quite universal: on one side or other about the temples or the occiput more or less unaffected portions of hair remain.

Events.—The disease does not in the strict sense spread beyond the scalp, but fresh patches arise, sometimes in the eyebrows, occasionally in the beard, more frequently on the skin of the neck and shoulders, and even on more distant parts. If left to itself, the course of the disease is extremely chronic, and shows little or no tendency to recovery—if the patient is a child—until the period of puberty is reached. It must not, however, be supposed that among neglected children in a village or a school where ringworm has invaded the community and scarcely a child has escaped, the disease constantly assumes the severe and inveterate character above described. A single bald patch may remain for months or years, or it may more or less completely recover and fresh patches go through the same series of changes; or, what is still more important to notice, the spores falling upon an unfavourable soil continue to multiply, and are thus a fresh source of contagion, but yet do not sufficiently interfere with the nutrition of the hair to produce obvious bald patches. In a family or school in which ringworm has appeared one may find evidence of its presence in the heads of children who are entirely without the characteristic bald patches.

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* They may be more easily detected by the naked eye if, as Dr Duckworth suggests, chloroform be first applied ('Brit. Med. Journ.,' November, 1873). This gives the affected hairs a dry, pale, brittle look, like that of burnt-up hay, apparently owing to its solvent power on the oily constituents of the hair. But this reaction is far from decisive alone.

Histology.—If one of the broken stumps of a ringworm patch be extracted with forceps and placed in a drop of liquor potassæ under a quarter inch objective it may be at once recognised in many cases by its absolute opacity. When less densely packed with spores or when soaking in potash has cleared it the condition is equally manifest by the complete destruction of all the normal histological characters of human hair. The cortex and medulla are undistinguishable, the surface is rough, the pigment no longer normally distributed, and the free end, instead of tapering to a point or being transversely cut off, is broken, slightly bulbous, ragged, or split into a sheath of fibres. A less degree of infection is recognised by a few spores in nucleus-like chains or a little branch of mycelium in the substance of hair apparently otherwise healthy. Dr Frederick Taylor has pointed out that the parasitic fungus does not invade the cutis itself, not even the follicle, and only slightly affects the epidermis adjacent. (Compare his paper, 'Med.-Chir. Trans.,' lxii, with Dr Thin's, ibid., vol. lxi.)

The spores differ from oil drops, with which they are often confounded, in the following particulars: first, they are uniform in size; secondly, they do not run together; thirdly, they are not perfectly spherical, but some at least perceptibly spheroidal or oval; next, they do not refract light so strongly, and though glistening and having a well-marked outline the centre is not so bright nor the circumference so broad and black; they occur in little groups or in chains; lastly, potash, instead of dissolving them by forming a soap as it does with oil drops, is powerless to affect their protoplasm, which is protected by a cell-wall and only serves to bring them out clearly by making the surrounding keratin and oily matter transparent. Ether is also without effect. Carmine and other staining agents act slowly, but in the end stain the cell. Often the most characteristic objects are not the extracted hairs but short broken fragments which are conveyed to the glass-slip with scales and *débris*.

Diagnosis.—The recognition of ringworm is in most cases sufficiently easy after a little experience, but we must remember that it may be masked by secondary impetigo as above described, also, that when of long standing it may produce patches of almost bald skin which may simulate the atrophic patches to be described hereafter (p. 709) as alopecia areata; and, thirdly, that the trichophyton may exist in hair which, as explained above, does not show the ordinary signs of ringworm visible without a microscope. In all doubtful cases, therefore, we must depend upon careful microscopical observation. This is particularly important when we have to decide whether the disease is cured or not. It is only by taking numerous specimens that we can assure ourselves of the fact. I have sometimes, where to the naked eye the ringworm was completely cured, hunted through a dozen slides without finding a single diseased hair, until in the last there was unmistakable evidence of the ringworm being still incompletely cured.

Ætiology.—I need not say that the only efficient cause of ringworm is the growth of the trichophyton tonsurans; and its almost universal spread under favourable circumstances shows that individual difference of soil has but little to do with it. What we do see are rather differences in the luxuriance of its growth, in the irritation it occasions, and in the obstinacy with which it clings to the affected scalp.

It has often been stated that ringworm occurs chiefly in pale thin children who are called "scrofulous" or "strumous," without any sign of enlarged glands or tubercle. I do not know of any evidence for this opinion. nor for its supposed predilection for light-haired, "lymphatic" children. One often sees ringworm in those who are the picture of rosy health. It occurs more frequently in light-haired children than others because most children in England have light hair, but it is common enough in brown hair, black hair, and red hair.

What is really important in its ætiology is that it is most frequent between the ages of three or four and puberty. It is rare in infants, particularly before the first dentition, and when present is usually cured without difficulty. This probably depends upon the less development of hair. Why ringworm of the scalp is rare in adults is extremely difficult to say. Not only do mothers and nurses with the disease among their children rarely take it, but when it does occur it is far more readily cured in adults. In children above ten or twelve years old it is easier of cure than in younger ones, and about fourteen or sixteen years of age it seldom gives trouble and sometimes disappears spontaneously. Occasionally I have seen obstinate cases in adults, but they are rare.

Ringworm is equally common in boys and girls.

Prognosis.—In infants and in adults ringworm of the scalp is a very manageable disease; in children, though the majority of cases may be cured with care and attention, it often proves obstinate, and now and then, in spite of the best available treatment, may persist for years and at last yield to advancing age alone. In a school or a family from a third to a half of the cases will be cured in a few weeks, a few of them by a few days' applition of the remedy. The majority of the rest will yield to persevering treatment in from three to six or eight months. A few only out of a large number will last beyond this time, and of them some at all events are pretty sure to prove inveterate.

Treatment.—The principle of treatment is the same as that of scabies. In both cases we know the cause of the disease, we know the natural history of the invading organism and the means of checking or destroying it. The difficulty in the case of ringworm is that most frequently before the case comes under our observation the fungus has already fixed itself deeply in the hair-sacs of the scalp, and it is extremely difficult to apply remedies to reach it. It is moreover protected by the epithelial scales which closely surround the hair bulb, and by the sebaceous and other products which block its mouth. We shall see that when the same parasitic growth invades the surface of the body its cure is easy.

So great are the practical difficulties of treating ringworm of the scalp that, although with perseverance and skill we can cure the vast majority of cases, and some of them rapidly as well as safely, yet everyone who has much experience in this disease must have met with cases which are so intractable that when after many months or even years they at last get well, it is to time and the increasing age of the patient that the cure is due.

Preparations of *mercury* are poisonous to all cryptogamic plants, to bacteria as well as to fungi, and probably the most poisonous is corrosive sublimate. A solution of perchloride of mercury in alcohol, two grains to the ounce, is sometimes rapidly effectual in curing recent cases of ringworm. It should, however, only be applied to separate patches, since there is at least one case on record in which its free use over a child's scalp produced (by some unusual accident in the application, or possibly some idiosyncrasy in the patient) absorption of the drug, and death by mercurial poisoning. Lotions, however, have the disadvantage of being repelled by the oily sebaceous infiltration of the natural and diseased structures of the scalp. We therefore usually prefer lard or vaseline as a vehicle, and in early cases of ringworm the white precipitate ointment (*ung. hydrarg. ammon.*) is often completely successful. It should be well rubbed into each patch morning and evening after thorough cleansing with hot soap and water and flannel. Instead of white precipitate ointment the *oleate of mercury** of the strength of one in twenty or one in thirty-five is effectual, and by many preferred to the older preparation. The 10 per cent. oleate is too strong unless applied to a very small patch in an older child.

Another parasiticide which has got into popular esteem is tincture of *iodine*. This also is sometimes effectual with recent cases.

There is, however, another method of destroying the fungus which is often found to be practically more efficacious. It consists in setting up a local inflammation the products of which effectually destroy the parasite. This plan is very applicable to the first stage of the disorder. If a mercurial application does not prove effectual within a few days, then, with older children, and especially on the first appearance in the family, it is probably better, after isolating the infected member, to attempt the immediate destruction of the fungus by exciting local inflammation. A stronger solution of iodine acts in this manner, but probably the most effectual and least painful application is the blistering fluid made of *cantharides*. The affected spot should first be shaved, including half an inch around it, and a circle of oil be drawn round the margin to prevent the blistering fluid from spreading. The pain of its application does not last long, and in many cases success is immediate and complete.

Too often, however, the fungus has already spread too far to be treated in this decisive manner, which is, I think, scarcely applicable except to recent cases with only a single diseased patch.

We will suppose that a child is brought to us with the disease established for several weeks with numerous bold rings, and perhaps with crusts and pustules from attempts to cure by various recent applications. The first step is to have the hair cut quite short over the whole of the scalp. Scabs and crusts must then be removed with the help of poultices and the whole surface made as clean as possible. We then see the real extent of the primary disease. It is often much less than it at first appears; secondary superficial dermatitis is readily cured and the diseased patches are soon ready for treatment. Sometimes we find no impetiginous crusts and little active inflammation, but scattered over the whole scalp small spots of ringworm and the apparently healthy hair between often furnishing evidence of infection. Under these circumstances the shortest and most effectual way is not merely to cut the hair short, but to shave it completely off. In inveterate cases it is much better to wait until the hair is removed, the crusts or scales got rid of, and the inflamed glands reduced before beginning active treatment. Meantime, the whole scalp should be well anointed morning and evening with carbolic oil one in fifteen or one in twenty, and the child's head covered with a linen cap both by night and by day. In this way no time is lost and the spread of the infection to other children is prevented. If without much active inflammation there is found considerable accumulation of dead epithelium, and especially when it takes the granular adherent character above described, this must be removed with potash, soap, or other alkaline applications. Dr Foulis ('Brit. Med. Journ,' 1885, vol. i, p. 536) has recommended for this purpose spirits of turpentine rubbed in until the child begins to feel it tingle, and then washed off with abundant warm water and carbolic soap. This is a rapid and effectual means, as I can testify, but it is only applicable when comparatively small patches are affected, and I would not recommend it for very young children.

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* Oleates were introduced by Dr Shoemaker, of Philadelphia (see 'Brit. Med. Journ.,' October, 1884).

When the way has thus been cleared for parasiticides we may in the slighter cases obtain good results by rubbing into each patch the white precipitate ointment as above recommended, anointing the intermediate surface with *carbolic oil.* In many cases, however, this proves inadequate, and we must then use stronger applications, although if the disease is extensive they must be applied only to a limited portion at a time. Equal parts of unguentum hydrarg. nitratis and sulphur ointment form an efficient and usually not too severe application. Dr Alder Smith, whose experience of ringworm at Christ's Hospital has been very large, recommends in obstinate cases a mixture of carbolic acid one part, citrine ointment one part, sulphur ointment one part. With children under ten two or three instead of one part of the sulphur ointment should be used to mitigate its effects and it will then cause no pain. Instead of carbolic oil (1 in 10 or 1 in 5) the carbolic glycerine of the British Pharmacopœia (1 in 4 or diluted to 1 in 8) is often preferred. It is preferable where lotions are being used. Another plan is to use carbolic oil (1 in 10) to the generally diseased surfaces and carbolic acid lotion to successive portions. I think, however, that this is apt to produce more pain and less certain curative effects than the compound ointments.

Some writers recommend *chrysophanic acid,* which is the efficient constituent of Goa powder, much used in the East Indies.* As I have said in speaking of its use in psoriasis, so here it is sometimes an extremely severe irritant, and always stains both the skin and linen unpleasantly. Chrysophanic acid has been tried, dissolved in chloroform, by Dr Alder Smith (seven grains to the ounce); and he recommends it in recent cases with only one or two spots as more successful than blistering. At the same time he uses a lotion of hyposulphite of soda two drachms to the ounce or of liquor sodæ chlorinatæ one part in eight.

Dr Crocker ('Lancet,' January 27th, 1877) reports careful and impartial trial of Goa powder in twenty cases of ringworm. Not more than eleven were only slightly improved after three months' treatment and only two were cured. Another objection to chrysophanic acid is that it is apt to get into a child's eyes, especially during the night.

A better application in every way is the ointment of *pyrogallic acid* which is much used against ringworm in Vienna.

Among the more severe applications is one introduced by Dr Costa, of Hanwell Central London Schools, and afterwards published in the 'Medical Times and Gazette,' vol. i, 1867, p. 34. This *Costa's paste* consists of two drachms of iodine dissolved in an ounce of colourless oil of tar, obtained by distillation from common tar and known as light oil of wood-tar or rectified spirit of tar, of sp. gr. 853 to 867. It is applied with a brush to the affected parts and forms a cake which separates at the end of a week or fortnight. (See a letter by Mr Martindale, in the 'British Medical Journal,' January 19th, 1880.) This was used by Dr Ringer at University College with success. Mr Morrant Baker, at St Bartholomew's, preferred iodine in the same proportion with creasote.

The most severe application is *croton oil,* which produces an artificial pustular dermatitis known as "kerion." A favourite ointment both in Germany and in France is that which is also used in the cure of scabies, a combination of sulphur with an alkali (Wilkinson's and Vlemingx's ointment). Hardy gives the formula: Carbonate of potash a quarter to half a gramme, sulphur one to one and a half gramme, lard thirty grammes.*

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* Chrysarobine is the trade name of Goa powder used at Bombay. The native name is araroba.

Instead of ointments or aqueous solutions the cure of ringworm has often been attempted with alcoholic lotions, but without marked success. Lately, however, Dr Cavafy ('Brit. Med. Jour.,' June 24th, 1882) has recommended a lotion composed of boracic acid, alcohol, and ether, in the following proportions: boracic acid twenty grains, ether one drachm, spiritus vini recti one ounce. The object, of course, is to dissolve the sebaceous material in the hair-sac and thus enable the boracic acid in solution to soak down to the spores which lurk there. This plan has been adopted and recommended by several writers of experience. I have myself tried it, having the lotion rubbed into the patches not less than four times a day. It is cleanly, painless, and sometimes effects a good and speedy cure. But, like all other applications, it has not unfrequently disappointed me.

Salicylic acid has also been employed in alcohol, ether, or chloroform. Corrosive sublimate may also be used in alcoholic solutions—two grains to the ounce.

If water lotions are preferred, sulphurous acid gas in solution (*Acidum sulphurosum* of the British Pharmacopœia) is one of the best parasiticides. It must be applied on pieces of rag to each patch. Or the hyposulphite of soda (two drachms to an ounce of water) may be used.

Thymol is another unirritating parasiticide which may be employed. It is soluble in alcohol and ether.

Dr Alder Smith recommends Barff's *boro-glycerine* as one of the best applications if the scalp is tender and sore, especially if impetigo is present.

Oleate of copper is an imitation of the old verdigris ointment, as that was of pennies laid in vinegar. It is of a bright green colour.

With oleates frequent washing is unnecessary and even undesirable. With solutions, whether in water, alcohol, chloroform, or ether, frequent washing with common or soft soap is absolutely necessary.

Is it desirable to aid the action of parasiticides by removing diseased hairs? This plan of *epilation* is generally carried out both at Paris and Vienna, and is adopted by many English physicians. Others believe that it is ineffectual. The fact is that to pull out all the diseased hairs over an extensive surface affected with ringworm is impossible even by a skilled manipulator. A certain number are sure to break off in the forceps, and still more are too short to be laid hold of. Moreover, the attempt is extremely tedious and painful, and the result insignificant. Where, however, a very small patch is for the first time seen, it is, I think, well to pull out at once all the hairs not only from the obviously diseased skin, but those in a small circle around, before applying acetic acid, blistering fluid, or any other agent by which we hope to destroy the parasite at once. Again, in chronic and extensive cases, removing loose hairs helps to prevent contagion, helps to clear the scalp, and also helps to ensure minute observation and care on the part of the nurse. It is therefore, I think, well to give her a pair of broad-tipped, well-roughened and weak-springed forceps, and to instruct her to remove every morning after washing the head as many hairs as seem to be loose, but not so as to cause the child pain.

Contagion.—While ringworm is under treatment the whole of the child's hair should be kept short, cut in fact as close to the head as may be; and this is probably as effectual as shaving. With girls a fringe of hair may be left round the forehead and behind the ears, so that, with a little skill, a cap may be worn during the day, and the child's appearance attract no attention out of doors. At night a linen cap should be used, and with younger children should be tied securely under the chin. Impervious coverings of gutta percha or oiled silk make the scalp hot and are unnecessary. There is no need for a quantity of ointment to be left on the scalp at night. The free application of carbolic oil, or carbolic glycerine, or oleate of mercury to the head is best undertaken in the morning. It is remarkable how very rarely mothers, nurses, or doctors, suffer from the most assiduous dressing of ringworm, but it is well to instruct the nurse to anoint her hands with carbolic oil each time she touches the child's scalp. With these precautions, and scrupulous avoidance of contact with caps, brushes, &c., it is possible for a child with ringworm to be treated and cured without removal from the family, but it is far better if the infected member or members can be separated from those who are healthy. If this is impracticable, they should sleep in separate bedrooms, and, if possible, meet only out of doors. It generally happens that in a family, while most of the cases are cured quickly, there remain one, or perhaps two extremely obstinate. These may, if necessary, be removed for the sake of treatment, but practically when the child has once been cured it is little liable to take the disease again, especially if the hair is kept short, if carbolic oil used as a pomade, and if the nurse (who if at all intelligent, will by this time be able to recognise the disorder) is careful to wash and inspect the scalp every week. It is obviously wrong for a child suffering from ringworm to be sent to school, for other children to be admitted to the house, or for its hair to be cut except by its own nurse.

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* Blistering is, in Dr Alder Smith's experience, rarely effectual by itself, and is almost always inferior, even in recent and circumscribed cases, to the application of a strong parasiticide.

The only proof of complete cure is the careful microscopical examination of the hairs not only from the previously diseased spots, but from the surrounding scalp. When the skin is itself perfectly healthy, and the hair which grows on it is soft and downy, with no broken stumps and black points showing under a lens, and when these good signs are associated with an absence of spores in the hairs examined, we may pronounce the child to be cured. It should, however, not be sent back to school for at least a fortnight after this, and should again be carefully examined before the risk of relapse can be considered past.

RINGWORM OF THE BODY.—*Tinea circinata.*—This affection, formerly called *Herpes circinatus,* occurs in the form of small rings with a red, papular, vesicular, or scaly margin. They are mostly confined to the face and neck, but are sometimes seen elsewhere on the trunk. They produce very little irritation.

The disease is contagious, and if scrapings from the ring are placed in potash under the microscope the mycelium and spores are apparent. There is more of the former, in proportion to the latter, than in ringworm of the scalp, and the fungus is not so readily seen, but when thoroughly soaked in potash it can always be discovered.

It often appears in children along with common ringworm, but may also be seen when the scalp is quite free from disease. It occasionally occurs in adults, especially in the form which will presently be described.

Burmese ringworm is the name given to what is described by the late Dr Tilbury Fox as nothing but a somewhat severe and troublesome form of *Tinea circinata.* (See his account of this and other exotic forms of ringworm in his work on 'Skin Diseases,' p. 541.)

Tinea marginata.—There is a form of tinea only observed in adults, and of which the parasitic nature was first recognised by Köbner. It was formerly called *eczema marginatum.*

Its distribution is very characteristic. Unlike all other forms of ringworm, it is symmetrical, and occurs only on the thighs, abdomen, perinæum, and buttocks. It begins, probably in all cases, with minute spots which rapidly form rings; but as these extend and coalesce, they produce gyrate figures, as above explained in the case of psoriasis, erythema and other disorders which spread at the edge. When a case comes before us, it has usually already assumed its characteristic aspect of a somewhat sinuous, broad, yellowish or brownish red, more or less inflamed band, which runs over the upper and inner part of each thigh, passes back to the fold of the nates or even as high as the sacrum, and then returns over the lower part of the abdomen or groin to the pubes. This curious distribution no doubt depends upon the mutual contact of the parts, and is aided by the warmth and perspiration which favour the growth of the fungus. The centrifugal spread is that of all forms of tinea, but the central parts are sooner free from the disease, the margin is more inflamed, and the duration much more chronic than in tinea of other parts of the body.

This curious affection is, so far as I know, confined to the male sex and adult age. It is most common in those whose occupation makes them sit for a long time together. It has thus been described in Germany as a disease almost peculiar to cobblers and cavalry soldiers. There is generally much irritation and, like all long-continued forms of dermatitis, pigmentation is produced not only in the growing margin, but also upon the inner exhausted surface.

The microscope demonstrates the same mycelium as is found in tinea circinata; but the disease may last for ten years or more, and when of very long standing it is often difficult and sometimes perhaps impossible to discover the parasite. Fortunately the aspect and distribution is sufficiently characteristic.

Treatment.—Tinea circinata is very easy of cure. White precipitate ointment or oleate of mercury, verdigris ointment (subacetate of copper two scruples, benzoated lard one ounce), tincture of iodine, boro-glycerine, sulphurous acid in solution—may all be employed with a certainty of speedy cure, which is in striking contrast with their action in ringworm of the scalp. In England there is no need for resorting to the more severe parasiticides, but in India Goa powder (chrysarobine, chrysophanic acid) was first introduced for so-called Burmese ringworm. It should certainly never be employed in the cases which come before us in this country.

Ringworm of the body is of course contagious, and may not only propagate itself but may lead to the development of tinea in the scalp. Its easy cure, however, renders precautions by isolation almost unnecessary.

Tinea marginata is, as above stated, very obstinate and difficult of cure. Sulphurous acid of the British Pharmacopœia freshly made, hyposulphite of soda (a drachm to the ounce), boracic acid (ten grains to an ounce of spirit), and corrosive sublimate (two grains to an ounce of water), may all be used with good effect.

In one very obstinate case, in which the patient, there seemed no doubt, had contracted the disease from a pair of knickerbockers which had been mended by a village tailor in Switzerland, I tried most of these remedies ineffectually for some months. At last I used pyrogallic acid ointment (half a drachm to the ounce), and the effect was so rapid and unmistakable that the patient rather blamed me for not using this cure before than thanked me for using it at last—and I am not sure that he was unreasonable.

According to a private communication with which I have been favoured by Dr Baldwin, of Rotherham, eczema marginatum of Hebra is not uncommon among artisans in the ironworks there who wear flannel next the skin and sweat much. He has found it yield to modified Wilkinson's ointment (p. 703).

ONYCHOMYCOSIS.—*Ringworm of the nails.*—It is happily very rare for tinea to attack the nails. When present it is usually a complication of ringworm of the scalp. Cases were recorded by Meissner in Vierordt's 'Archiv,' by Virchow and by Bazin, as early as 1853. It has been carefully described and the microscopic appearance figured by Neumann ('Hautkankheiten,' p. 347, figs. 48 and 49), by Dr Purser ('Dubl. Quart. Jour.,' Nov., 1865), and by Dr Fagge in the 'Guy's Hospital Reports' (3rd series, vol. xv, p. 553, and 'Clinical Transactions,' vol. i, p. 77). I have myself seen only one case. The nails become yellowish and brittle, but not rough as when affected by eczema or psoriasis. The fungus may be that of favus (*achorion*), or of common ringworm (*tricophyton*). Good models of onychomycosis due to both parasites will be found in the Guy's Hospital Museum, Nos. 536 and 537.

It is the most obstinate of all forms of ringworm, and will often persist during the whole of childhood, and only disappear spontaneously after puberty.

The *treatment* recommended is scraping the affected nail, softening it with alkalies, and when other means fail, complete removal, together with the sedulous application of sulphurous acid or hyposulphite of soda; but I know of one case in which an eminent dermatologist adopted this method in addition to every other possible parasiticide treatment, without curing the disease.

FAVUS.—*Tinea favosa—Porrigo lupinosa.*—This is a rare affection of the scalp and body, due to the presence of a fungus named *Achorion Schönleini.*

The disease was recognised and named by Bateman, and was figured by Alibert. But it was not till 1839 that Schönlein published in Müller's 'Archiv' the discovery that the yellow crusts of favus were neither pustular nor sebaceous, but were composed of the mycelium and conidia of a parasitic fungus. This discovery preceded that of Malmsten and Grube above mentioned (pp. 691, 698, 709), and therefore Schönlein has the great merit of opening the whole chapter of cutaneous mycology.

In its earliest stage favus is probably undistinguishable from common ringworm, but very soon a characteristic flat, round, yellow object is seen, depressed in the middle, opaque, adherent, and perfectly dry. Its colour has been compared to a honeycomb (*favus*), and its shape to the disc of a lupine seed. The sight of a single case of the disease or of such models as Nos. 523—527 in the Guy's Hospital Museum, or even of a well-executed drawing, is sufficient to enable anyone to recognise favus.

The individual crusts amplify, coalesce, and form thick rugged, porous, yellowish masses, resembling somewhat the rind of old wormeaten cheese. They have a characteristic mouldy odour which resembles that of mice.

The disease may affect any part of the body, but is particularly severe upon the scalp, where it destroys the hair-sacs and often produces complete baldness. It is, as above stated, rare in England, but is less so in Germany and comparatively common in France. It appears also to be not infrequent in Scotland. Mr Hutchinson published forty-four cases with instructive remarks upon the disease in the 'Med. Times and Gazette' for 1859 (vol. ii, p. 553).

Favus has been recorded by Dr Purser, of Dublin, in a cat (1866), and by St Cyr in rabbits and mice ('Ann. de Dermatologie et Syphilis,' 1869), quoted by Dr. Fox ('Skin Diseases,' p. 431).

The *treatment* is unsatisfactory. Ordinary parasiticides produce improvement, and if perseveringly employed, apparent cure; but relapse is almost sure to occur. The old French treatment of epilation by a cap of pitch-plaister applied to the head and then torn off is no more effectual than less barbarous methods, but epilation is probably necessary for even a temporary cure. Several cases of this remarkable disease are described by Dr Fagge in the 'Guy's Reports' for 1870 (p. 354), where more than one apparent cure by epilation is recorded.

It will be convenient to consider in this chapter the remaining affections of the hair, some of which were formerly confounded with ringworm and are still liable to be mistaken for it.

Alopecia.—Baldness, or loss of hair, when not the result of the presence of *Trichophyton tonsurans,* is the immediate consequence of atrophy of the hair-bulbs, which occasions the premature fall of the hairs from the follicles. When this is only partial and followed by fresh growth of weak hair, the result is thinness or partial baldness, but when the hair-sac is no longer capable of producing a fresh hair, complete alopecia results.

Although a senile change, baldness cannot be considered strictly physiological; for it is often absent even to advanced age in men, it is usually absent and rarely complete in women at any age, and it sometimes occurs very early without any other signs of senile decay. In these cases it is frequently hereditary, but by no means constantly so.

The atrophy of the hair-sacs certainly does not depend upon general deficiency of healthy nutrition, nor upon locally deficient supply of blood. It is not accompanied by anæsthesia, by numbness, or by any other evidence whatever of nervous disorder, so that to ascribe alopecia, whether premature or not, to "vascular" or "neurotrophic disturbance," is arbitrary, and does not advance our knowledge. It has been asserted that adhesions of the pericranium, and particularly want of mobility of the aponeurosis of the occipito-frontalis, produces alopecia, but I have seen many instances which disproved the assertion. Neither wearing tight hats, nor going without hats, nor wearing turbans indoors, nor exposure to the sun—nor gout, nor scrofula, nor intemperance, nor abstinence—none of these will in the least explain either senile or premature baldness, for each supposed cause is refuted on examination.

Alopecia of this quasi-physiological character begins usually in the frontal region, sometimes at the central point at the back of the head from which the hair falls forward, backward, and laterally, and not unfrequently, in both regions at once. There is often seborrhœa sicca or a slight degree of pityriasis which precedes and accompanies baldness; but if this is the cause, the thinness of hair can be cured by restoring the skin to a healthy condition, and even if neglected it does not go on to complete alopecia. Moreover, in many cases both of senile and premature baldness the skin is healthy throughout. When the hair has fallen off from the mid region of the scalp, the process almost always ceases, and that on the temples behind the ears and on the occiput persists without change. This ordinary alopecia, moreover, never affects the beard, the eyebrows, or other parts of the body.

Many attempts are from time to time made, not only by tradesmen but by physicians, to check the loss of hair or to restore it. They consist either in applying stimulants of which cantharides is usually the basis, or in shampooing and manipulating the scalp. It is very rarely that these attempts have even partial success. A process introduced a few years ago by Dr Pincus, of Berlin, promised better, but has not in the sequel fulfilled the expectations of its author.

Alopecia as the result of febrile diseases.—Although this often proves the first step of ordinary baldness, yet it is distinguished therefrom by its affecting both sexes and all ages, by the fall of hair not being confined to any region of the scalp, and by its thinning rather than completely stripping the surface affected. Moreover, it is not only secondary in origin, but usually passes away of itself after convalescence, instead of being practically incurable either by nature or by art.

Syphilitic baldness agrees in these characters, and its frequency, apart from any other affection of the scalp, as well as its early appearance, likewise point to its ætiology as a febrile alopecia.

Area.—*Alopecia areata* (Sauvages), *Area Celsi*, *Porrigo decalvans*, (Willan), *Teigne pelade* (Bazin), or *Tinea decalvans.*—So peculiar is the appearance of this disease, that it is less needful to insist upon its distinction from other kinds of alopecia, than upon the fact that it is a true alopecia, anatomically identical with the other forms of atrophy of the hair though differing in its origin and course.

The first of the above titles appears to be best, since it is distinguishing and is generally accepted. Celsus did not particularly describe this variety of baldness, but applied the word "area" ("a bare space," *locus sine ædificio*) to any form of baldness, distinguishing ἀλωπηκία and ὀφίασις as varieties. The "porrigo" of Willan meant any eruption of the scalp, including true ringworm and impetigo or pustular dermatitis, and the term is now almost out of use. The appellation Tinea or Teigne depends upon the erroneous doctrine of the parasitic nature of the disease.

On this point I am entirely in accord with most modern dermatologists. I have many times sought for a fungus and have never found the smallest evidence of its presence, with one single exception. This occurred nearly fifteen years ago when I was working under the late Professor Hebra. In one of his patients suffering from area I discovered some spores and scanty mycelium in one of the neighbouring hairs. I showed it to the professor and he told me that he had never seen it before. He doubted whether its occurrence was more than accidental, and with my present experience I doubt it also. Hebra himself believed at one time in the statement of Gruby that the disease was parasitic, but had long changed his opinion, and I can only share in the surprise expressed by Dr Kaposi (Hebra's 'Hautkrankheiten,' ii, p. 149, note) that the author is associated with Bazin as a supporter of the parasitic nature of area by his disciple Dr Neumann ('Lehrbuch der Hautkrankheiten,' p. 297). It is possible that the single observation of Gruby in 1843 ('Comptes Rendus,' xvii), which gave rise to the question, was made upon a case of true ringworm. Neumann, who has no doubt that area is not parasitic, once, like myself, found some spores in a case of the disease, but like myself doubts rather the significance of a single observation than the accumulated testimony of his own and others' experience. In fact M. Bazin's statements (and those made recently by Malassez and by Eichhorst) are, I believe, the only ones which rest on large experience and assert the presence of a fungus. The French dermatologists call many cases "pelade" or "teigne pelade" which in England or Germany would be regarded as true ringworm in its later stages. In Mr Hardy's lectures it is not difficult to recognise in the swelling, irritation, and desquamation of the skin which he describes in pelade,* the characters of ringworm. I never saw at the hospital of St Louis an attempt to demonstrate the presence of spores in what we should call a genuine case of area.

Apart from the microscopic evidence, the naked-eye appearance and natural history of the disease would almost disprove the parasitic hypothesis. The hairs around the affected spot are not swollen at the root, nor brittle in the shaft, but are merely atrophied like normal hairs which are ready to fall out. There is no evidence of local irritation in the hair-sac. The disease above all is not contagious, at least as we observe it in England, and it is not curable by anti-parasitic treatment.

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* 'Leçons sur les Maladies de la Peau,' 2me partie, pp. 179—184.

Dr Thin ('Proc. R. Soc.,' 1881, No, 217) has figured minute schizomycetes which he calls *Bacterium decalvans*, but which are rounded rather than rod-like, and probably identical with those described by Dr v. Sehlen in 'Virchow's Archiv.' If this is of ætiological importance, it does not, of course, make area a true tinea.

Another theory is that area is a tropho-neurosis, for which I can see no sufficient evidence. The subject was discussed in the International Medical Congress of 1881, by Hardy and Vidal, Kaposi, Liveing, and others (vol. iii, p. 158).

Area is certainly more common in children and young adults than after thirty. It seems to affect both sexes nearly equally. In eighty-one cases of mine, fifty were men. In many cases it probably would recover of itself, but I believe that recovery is often hastened if not brought about by treatment.

This consists in local irritants, and, when necessary, internal corroborants. I usually begin with a lotion containing ʒiss to ʒij of acetum cantharidis to a pint of water. This will often cause slight erythema in children, but in adults and in many children we may increase the strength to two, three, or four drachms with advantage, letting the irritation subside whenever it goes beyond redness on to excitation. A mild and often efficient application is *linimentum myristicæ* (one part of the expressed oil to three of olive oil) which I learnt from Sir William Gull when he had charge of the department of skin diseases at Guy's Hospital. With brown hair, the *unguentum iodi* of the Pharmacopœia is a useful application.

Area occurs in persons of all degrees of health, complexion, and temperature, but if the patient is pale and thin, steel is certainly useful; and I often prescribe bark or cod-liver oil, but only when indicated by some other symptom than the bald patches.

I have seen two or three instances of a second attack of area appear after the first had been completely cured and an interval of time had elapsed.

Universal alopecia.—There are some cases of complete and rapid loss of hair which are neither senile, syphilitic, nor febrile, and which, I believe, cannot be classed as examples of area. They are distinguished first by the hair falling off almost simultaneously from the whole of the scalp, not gradually from certain regions as in ordinary baldness, nor by the confluence of separate patches as in area; secondly, by the baldness not being confined to the scalp (nor even to the scalp and beard or eyebrows as I have seen it in area), but affecting the whole of the body; thirdly, by its not following an illness.

In one case of this kind the patient was a young man in robust health and wearing a full beard. Without any assignable cause he lost the whole of the hair of his body in a very short space of time. I have notes of eight other cases, two in women, and comparing these with the most rapidly spreading cases of alopecia areata, I think they may be fairly distinguished as constituting a separate form of baldness.

This universal alopecia occurs in both sexes, always beginning in adult life, and always in young adults. It is quite incurable.

We may at present distinguish these somewhat rare cases of *alopecia universalis acquisita* from the still rarer cases of *congenital alopecia.* In these, the nails, as well as the hair, are affected; and like other deficiencies of development, the condition may be hereditary. Such cases are comparable with congenital ichthyosis, especially in such marked examples as the "porcupine boy," and still more closely with the "hairy family" of Burma, and the blue and hairless horse exhibited a few years ago in this country.

A striking series of examples of this form of baldness occurred five years ago in this hospital under Dr Fagge. It is remarkable that the development, both of hair and nails, was tardy and imperfect, but not absolutely deficient. The italic letters denote the female sex.

F. Born without hair or nails. Hair began to grow when he was about twenty-three years of age, and at thirty he had a full head of hair. The finger-nails also grew after puberty, but were always ill formed, and he never had toe-nails.

F. Normal.

B. 1. Born without nails or hair. The former appeared while teething, the latter when she was ten years old.

n. Born without hair and nails. None yet grown.

B. 2. Born with hair but without nails. Died, aged seven.

B. 3. Born without hair or nails. Died, aged five months.

B. *B.*, 4—9. Born with normal hair and nails.

B. 10. Born partly bald with ill-formed nails—was under Dr Owen Rees, when a boy. He is now twenty-two and has a fair head of hair, but his nails are not good.

The patient herself, then nineteen years old, the eleventh and youngest of this large family, was born without hair or nails. She had in 1876 only thin lanugo on the scalp and imperfect nails.

TRICHOCLASIA (*Wilson*).—A singular disease of the hair, which has been described under this title, and also as *fragilitas crinium* and *Trichorrhexis nodosa* (Kaposi) is characterised by each hair dilating at intervals and breaking at these enlarged points. The dilated node consists of separated cortical fibres which look very much like the splitting and enlargement of a cane when broken across, and the air which enters between the fibres makes them appear white by reflected light. They have thus a superficial resemblance to the ova of pediculi.

It is almost always confined to the beard, is non-contagious and non-parasitic.

It was described by Devergie as "trichoptylose," and subsequently by Beigel and by Wilks. I have seen three or four cases of it, one of which I figured in the 'Pathological Transactions,' for 1879 (p. 439), where I gave references to the scanty literature of the subject.*

This is apparently quite distinct from a parasitic affection of the hair, known as "Piedra" from its stony hardness, which occurs in the hair of the scalp among women only in Central and South America, and has been described by several French writers and by Mr Malcolm Morris in the same volume of the 'Pathological Transactions' (p. 439).

TINEA VERSICOLOR.—This affection, described by Willan as *Pityriasis versicolor*, was formerly named *maculæ hepaticæ*, a translation of the vernacular German name *Leberflechte* (liver spots, *chaleur de foie*). Another name still often applied is cloasma, but this is better reserved for true maculæ produced by pigment.

In 1846 Eichstädt published in Froriep's 'Journal' the discovery that this affection is due to the presence of a fungus. It is worth noticing, now that its real nature is understood, Bateman's remark, that "the causes of this pityriasis are not well ascertained; fruit, mushrooms, sudden alternations of heat and cold, violent exercise with flannel next to the skin, have been mentioned as probable causes. The most extensive eruption I have seen occurred in a Custom-house officer after drinking spirits freely during a day of fasting on the Thames."

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* See also a valuable paper, with fuller references, by Dr T. C. Fox, in the 'Lancet,' Dec. 7th, 1878, and Hans v. Hebra, loc. cit., p. 391.

Tinea versicolor occurs as yellowish-brown spots of various shades, scarcely rising above the level of the skin, and yielding a branny or furfuraceous desquamation when scratched. The spots vary from a pin's head to several inches in diameter. As they multiply and coalesce, they form large patches and the advancing border rings, which when united produce the gyrate or serpentine outline before described as the result of this mode of development of an eruption. It is rare, however, to see such perfect rings as in tinea circinata, and the central parts seldom completely recover and almost always appear more or less discoloured.

The *distribution* of this affection is very characteristic. In the great majority of cases it occupies the chest, often spreads to the abdomen, and is frequently seen on the back, especially between the shoulders. It may overspread the whole trunk, but rarely descends below the waist or ascends above the neck. Occasionally a patch or two may be also found on the border of the axilla and on the soft skin of the inner part of the arm and on the bend of the elbow. Even when the abdomen is not affected it is common to find this form of tinea on the inner side of the thigh, whence in males it is apt to spread to the scrotum. We may say, therefore, that the affection never occurs upon parts which are exposed to the air, and that its favourite seat is on skin which is the most protected and the most constantly warm and moist.

On scraping some of the surface and putting the scales in a drop of potash under a microscope both spores and mycelium can be seen without difficulty. The spores of *Microsporon furfurans* are somewhat larger than those of *Trichophyton tonsurans* and occur in considerable heaps, which are surrounded by mycelium threads. The presence of the fungus is of course the decisive point of diagnosis; but with a little experience the colour, the branny desquamation, and the locality of this affection are sufficiently characteristic.

I have sometimes found patients complain a good deal of the irritation occasioned by tinea versicolor, and it may be accompanied, especially in hot weather, by slight erythematous dermatitis or urticaria, as the result of scratching. Most frequently, however, it produces no symptoms whatever and is either discovered accidentally or is only regarded as a disfigurement or uncleanliness. The superficial layers of the epidermis are alone affected by the parasite.

The *cause* of this curious affection, or rather of the fungous growth on which it depends, is quite unknown. It is remarkable that it seldom, if ever, occurs in children, and is rare after middle age. It is most often seen in men between twenty and forty, but may also be observed in women, especially under the fold of the mamma. Although the fungus has been proved by experiment to be capable of transmission by direct inoculation, the disease is not practically contagious, or if at all, to a very small degree.

Treatment.—If left alone tinea versicolor continues indefinitely, but it may be readily removed by any of the milder parasiticides. After thorough washing with hot soap and water, or, if a rapid cure is desired, with soft soap, the affected parts must be well rubbed with oleate of mercury or unguentum æruginis (℈ij ad ℥j), or, if preferred, sulphurous acid or hyposulphite of soda (ʒj ad ℥j) may be applied in watery solution.

Tinea rosea (?)—*Pityriasis rosea.*—This affection was first described by Gibert as an acute centrifugal erythema of the trunk. Bazin called it *P. rubra maculata et circinata.* Vidal regards it as parasitic ('Trans. Intern. Med. Congr.,' vol. iii, p. 133). German writers consider it a variety of ringworm of the body.

CHRONIC DEEP INFLAMMATIONS AND HYPERTROPHIES.

Deep dermatitis—its definition—its relation to eczema and other forms of superficial dermatitis—to hypertrophy—and to new growths.

GUTTA ROSEA.—*Origin and development—Localities—Causes and pathology—Relation to dyspepsia—to drink—to ovarian irritation—Treatment.*

EPIDERMIC HYPERTROPHIES.—*Callosities and corns—Leucoplacia lingualis et buccalis—Warts—Condylomata and mucous patches.*

ICHTHYOSIS.—*Anatomy—Varieties—Xerodermia—Treatment—Horns—Ichthyosis intra-uterina.*

SCLERODERMIA.—*History—Description—Distribution—Histology—Diagnosis—Treatment—Sclerema neonatorum—Linear atrophy.*

ELEPHANTIASIS.—*Nomenclature—Anatomy—Pathology—Relation to chyluria and filaria sanguinis—Clinical characters.*

As stated in the introductory chapter, the great majority of affections of the skin consist pathologically in superficial inflammation, that is to say, inflammation which affects only the papillary layer of the cutis and the Malpighian layer of the epidermis, with the resulting change in the cuticle. In no form of this superficial dermatitis are the papillæ destroyed; and no scars result. We have now to speak of a far less frequent kind of inflammation of the skin which involves, if it does not originate in, the deep layer of the cutis, which destroys the papillæ, which spreads from the skin proper to the subcutaneous connective or adipose tissue, and which after recovery leaves scars behind. Eczema, psoriasis, and their allies, scabies, the erythematous eruptions, and the parasitic affections—are all, in the sense in which the word is here used, superficial; and however severe and protracted their course, when cured, they leave either no trace behind or only a pigment spot.

It is true that when inflammation occupies the deep sacs of the hairs and the sebaceous glands a cicatrix is not unfrequently the result.

Thus acne in its severe forms leaves scars behind varying from mere white spots, very slightly depressed and otherwise inconspicuous, mere local atrophies, up to the hypertrophied scars which sometimes simulate cheloid. The same applies to sycosis, though obvious scarring is less frequent. Some other pustular diseases destroy the papillæ and thus produce scars. This never occurs with true impetigo (which is one of its pathological as well as diagnostic characters) nor with the pustules of scabies or bullæ of pemphigus; but variola, when unmodified by vaccination, almost always leaves indelible traces of its presence—either deep-pitted, depressed, white scars or more extensive and hypertrophied puckering. The same is true, though less constantly, of varicella, and the deeply pitted cicatrix is the well-known mark of successful vaccination. Lastly, the pustules of zona very often (though by no means constantly) leave more or less marked cicatrices and sometimes, especially upon the forehead, these are deep and indelible.

But beside these deep pustules, we meet with inflammation of the skin which, uniformly and over large surfaces, penetrates below the papillæ and affects the whole thickness of the integument, together with the subcutaneous tissue. Such *deep dermatitis* is usually chronic in course; or, if it shows acute characters, they are repeated again and again, without any tendency for the malady to come to a natural end. Such recurrent subacute diseases become practically chronic, as we see in the case of inflammations of the bronchial tubes, of the eye, and of the colon.

Like other chronic inflammations, those of the skin show in many cases little of the classical signs and are unattended with fever; moreover, the exudation is never purulent, but if œdematous gradually assumes the characters of *œdema durum*, if congestive gradually assumes those of hypertrophy. The inflammatory cells or corpuscles, instead of dying and undergoing transformation into pus-cells, become organised into connective-tissue corpuscles, and gradually form fibres. Thus chronic inflammations are closely related to, and often undistinguishable from, *hypertrophy* in the humbler stages of that process, hyperplasia of the connective tissues.

Again, chronic inflammation is apt to lose the uniform and characteristic qualities which distinguish the catarrhal, adhesive, and suppurative forms of acute inflammation as described in the first volume (p. 56). Thus chronic catarrhal broncho-pneumonia is apt to assume a *caseous* form and ultimately to lead to the new growths which we call tubercle. In the present work Dr Fagge has advocated the inflammatory nature of tubercular phthisis (p. 68), but phthisis does not follow acute pneumonia. Again, chronic inflammation of the urinary tract often ends by becoming caseous and tubercular.

Again, the continued irritation which gives rise to inflammation and thickening of the mucous membrane of the tongue, the lips, the pylorus, or the rectum may in time, by almost imperceptible stages, pass into a *new growth*, perhaps of the most markedly "heterologous" and malignant kind. Warts and other innocent growths, condylomata and syphilitic nodes also arise from and are complicated with chronic inflammation and hypertrophy.

It is therefore pathologically justifiable to associate with chronic deep inflammations of the skin, hypertrophies, tubercle and new growths; and this arrangement we propose to follow.

The only important instance of *acute* deep dermatitis is that afforded by erysipelas, which has been already treated as a specific disease in the first volume. The deep and acute inflammations which result from burns and other injuries are best studied in surgical text-books.

GUTTA ROSEA.—*Acne rosacea, Couperose.*—This affection in its more obvious forms is well known beyond professional circles. A classical instance of it provoked the well-known descriptions by the English knight and the Welsh squire: "the lanthorn in the poop," "an everlasting bonfire light;" "his face all bubucles and whelkes and knobs and flames of fire," "sometimes blue and sometimes red."

Short of Bardolph's degree, it is not uncommon to see gutta rosea, and it is far from being always the result of intemperance.

Course.—The affection begins with slight erythematous redness, usually of the tip of the nose, occurring after food and combined with local irritation; the heat and itching are felt by the patient, the redness and even slight swelling are visible. It passes off quickly and perhaps may not return for days or weeks, but gradually becomes more frequent, until it is at last habitual.

The next step is for the congestive vessels to fail to recover themselves in the intervals between the successive states of hyperæmia. What was a recurrent subacute erythema becomes a chronic dermatitis with exacerbations. Frequently-recurrent œdema has moreover ended in hypertrophy, so that the skin and subcutaneous tissue of the affected part are swollen and thickened. Some of the veins from habitual distension become varicose and remain visible as red tortuous lines. The sebaceous glands are apt to be obstructed or to inflame without obstruction, and pustules resembling those of inflamed acne result. Hence the common name "acne rosacea." But there are no precedent comedones, and the distribution, ætiology, and entire natural history of the disease are distinct from those of acne.

Hypertrophy may go on until great pendulous masses of thick skin, with the scars of past pustules and abundant fibrous tissue, form hideous excrescences upon the nose, growing either from the tip, from the alæ, or from the septum.

Distribution.—By far the most frequent seat of gutta rosea is the nose, but it is not the only one. In persons in whom this feature is characteristically affected we usually find large red pimples with inflamed base and chronic course upon the cheeks, the chin, and other parts of the face. When the nose is only slightly affected and the rest of the face decidedly, the appearance is very different from that of the hypertrophied form above described when confined to the nose, but the anatomical condition is essentially the same, and every gradation between the two forms may be observed. Beyond the face, similar recurrent erythema, with more or less of hypertrophy, may be seen in the lobes of the ears, although here it is very rare to see pimples or pustules. We never find a corresponding condition of the shoulders or chest, as we do in acne.

Causes and pathology.—I believe that gutta rosea is no less distinctive in its ætiology than in its anatomy and distribution. It is essentially an erythema, or rather the result of frequently-recurring erythema. At p. 669 I have described the congestive erythema with œdema and hypertrophy which affects the extremities when from any cause the circulation in them is inactive. The difference between chilblains and their allies and gutta rosea is anatomically that the former is a passive cold venous congestion, and the latter an active, hot, arterial hyperæmia. Like other erythemata, gutta rosea is symptomatic (p. 668); it is never the result of local irritants, it always depends upon reflex inhibition of vaso-motor nerves causing active congestion. We saw that the origin of this reflex action is in some of the most marked forms of erythema irritation of the primæ viæ by poisons, drugs, or food. Gutta rosea is no exception to this rule; it is almost always the result of gastric irritation.

Common notoriety affixes the stigma of drink to the possession of a nose like Bardolph's, but it would be no less unjust than uncharitable to assume this as the necessary cause. All we can say is that the excessive use of alcohol produces most frequently and most readily the gastric irritation which leads to gutta rosea, but marked examples of the disease may be seen in persons of habitual temperance, and even in total abstainers. In women, especially at the period of the menopause, there is very apt to be a form of dyspepsia which leads to flushings of the face, not only after every meal, but in the worst cases, upon putting the first morsel of food into the stomach; these flushings are felt by the patient and cause great distress. This frequently-recurring hyperæmia leads to habitual congestion, pimples, and more or less hypertrophy, although in these cases the type is more often that of diffused redness with pimples scattered over the face than the local hypertrophy of the nose. This, however, is occasionally seen, just as the diffused pimply redness is often the result of tippling.

The only cause for gutta rosea beside alcoholic or non-alcoholic dyspepsia is uterine, or rather ovarian disturbance. The frequency with which disease occurs when menstruation is becoming irregular before it finally ceases seems to make this probable. In many cases of gutta rosea the affection is decidedly worse at the menstrual periods, and is associated with dysmenorrhœa. Considering, however, the great number of cases of menstrual disturbance in which no such effect is produced, and the extreme rarity of gutta rosea even in the worst cases of dysmenorrhœa in young women, as well as the frequency of what may be called climacteric dyspepsia, I am disposed to think that in almost every case gastric irritation is the exciting cause of the disease, and that the monthly exacerbations which undoubtedly occur in certain cases are due rather to direct physiological vascular excitation at that time than to morbid irritation of a reflex kind.

Gutta rosea is not produced by the most frequent kind of dyspepsia, that of young adults; it is rare before the age of forty, even in persons who drink freely; it is often combined with acute dyspepsia, but one should, I think, consider it rather as the result of dyspepsia which, like the gout, is produced by over-feeding and over-drinking rather than directly connected with the presence of urate of soda. Gutta rosea is very far from being confined to the male sex, though the most typical cases from alcohol are of course more frequent in men.

Treatment.—The true treatment of this, as of every other disease, depends upon recognising its pathology and origin. The first indication is to remove the gastric irritation which is almost always present, to discover if possible its cause, whether in excess of food, in imperfect and hasty mastication, or in some particular article which acts as a poison. Salt meat, spices, pickles, melted fats, and sauces, any of these may prove to be the offender; but most frequent of all are wine, beer, or spirits. If we fail to discover the cause of the dyspepsia we may yet do good, apart from diet, by the exhibition of small doses of soda with rhubarb and columba, or when gastralgia is marked, by ten grains of subnitrate of bismuth as a powder either before or after meals, to which five grains of carbonate of soda may be added if there is obvious acidity. Gentle laxatives are often desirable and occasional doses of blue pill. In some cases euonymin is particularly valuable, taken in doses of two or three grains every other night; in others a dinner-pill of colchicum and nux vomica with extract of aloes is found useful. Locally, astringent washes, like Goulard lotion, are useful and pleasant; flexible collodion may also be painted over the congested parts at bedtime. In advanced cases, scarification by innumerable punctures with a lancet is sometimes efficacious. Successful cases have been reported by Mr Squire and Dr Stowers. When the hypertrophied masses are considerable they can only be removed by the knife.

EPIDERMIC HYPERTROPHIES.—It was observed by John Hunter that internal pressure produces atrophy, as when a tumour or aneurysm presses upon vertebræ; but that external pressure produces hypertrophy, as in pressure upon the skin of a labourer's hand: or rather continuous pressure produces absorption and atrophy, intermittent pressure produces hypertrophy.

When pressure is continuously applied, as to a lady's foot in China while still growing, atrophy takes place with only moderate distortion of the bones and without thickening of the skin; but when it is applied only while walking, as by the narrow-toed and high-heeled shoes of a European lady, there ensues, along with a certain amount of distortion, hypertrophy or thickening of the prominent parts of the skin. This is usually accompanied with a chronic deep dermatitis whereby the papillæ are affected and a new growth forms or occasionally a deep bursa results. These products, in which chronic inflammation, hypertrophy and tumour are seen at their point of junction, we know by the names of corn and bunion.

When without unnatural pressure or distortion, the hand or foot or any other part is exposed to intermittent pressure, the result is something short of this. It is a pure hypertrophy affecting only the epidermis (*callositas*, *tyloma*). Such is the case in the thickened skin of the ball of the foot and the heel in adults, and of the palm of the hand in all who do manual labour. In children the thickening is but very slight, probably an inherited character, since we find it in all plantigrade animals. In adults the degree of it varies with the habits of the individual. This most purely physiological form is seen in those races who go barefoot, for wherever shoes are worn there is a chance of corns appearing even on the sole of the foot. Precisely similar callosities appear in the middle of the palm in workmen who use screwdrivers, gimlets, and augurs, in the cleft between the finger and the thumb in shoemakers and others who habitually pass a strap or cord in this position, over the patella in those who frequently kneel, and on the back of the neck, especially over the sixth vertebra, in those who carry burdens on their shoulders, as may be often observed in railway porters.

In such a callosity it will be found on section that the horny layer or cuticle is enormously increased, the Malpighian layer slightly, if at all, and the cutis vera quite unaffected. Hence these callosities appear lighter than the rest of the skin in negroes.

The corn (*clavus*), as was first shown by the anatomical researches of Gustav Simon, consists of a diseased growth of the horny cuticle into subjacent living Malpighian and papillary layers. The horny downgrowth is of a more or less conical shape and causes atrophy of the immediately adjacent papillæ, but at the same time a thickened layer of cutis forms around by true chronic inflammation. Here the cuticle is but slightly thickened and not hard as in the central part, and the papillæ become gradually hypertrophied. Occasionally the original central hardening appears never to take place, especially in the soft parts of the skin between the toes, which are continually in contact and moistened with perspiration. The result is what is known as a "soft corn;" or there may be a mere horny plug pressing on the skin beneath, but without exciting inflammation around. This occurs most frequently on the naturally thickened skin of the ball of the great toe or heel. In this case the resulting pain is that of an occasional sharp prick, when the sharp, hard, horny plug is suddenly driven home by accidental pressure. This is very different from the continual, tender, wearing, and disabling pain of a clavus mollis. The commonest kind of corn, partaking of both characters, combines the discomfort of each. When a cyst or bursa forms beneath the corn and increases so as to become obvious it is called a bunion. A small cyst is often to be found beneath an ordinary corn of old standing and large dimensions, but the large cysts almost always form over the metatarsal pharyngeal joint of the great toe when this has been rendered artificially prominent by the distortion of short, narrow-toed, and high-heeled shoes. The bursa from time to time inflames and the tension then occasions severe pain, although suppuration is rare.

Treatment.—The proper treatment of corns is prevention. Children's shoes should be made low in the heel, broad in the tread, perfectly straight on the inner side, and each shoe markedly unsymmetrical. In measuring for shoes or for making a last, one should not sit but stand, so that the weight of the body may expand the foot into the natural shape and size which it then assumes. In a perfect covering for the foot similar expansion is afforded by the elasticity of the upper leather and the yielding of a thick and soft stocking, but the sole should slightly project so as to equal the largest length and width of the foot. Even in adult life the trouble of insisting upon boots being properly made is well repaid by the increased comfort and ability to walk, and the disappearance of acquired corns and distortions. In bad cases it is well for the patient to wear stockings with divided toes like a glove, and to have a stout vertical piece of leather fixed so as to separate the great toe from the rest and to press it outwards into its natural position, as was advocated by the late Aston Key.

Beside removing the thickened epidermis and extracting the conical plug of hard keratin from time to time, relief may be obtained by treatment with salicylic acid, 2 per cent., mixed either with mutton suet, forming the ointment in use in the German army, or in the stronger proportion of five or ten grains to the ounce of vaseline, or as a plaister, as recommended by Dr Unna, of Hamburg. Where, in addition to soft corns, the whole foot is tender and painful, the remedy consists, first, in large and low shoes so as to diminish the heat and moisture; secondly, in thick and loose knitted stockings, which are at once absorbent and pervious; and, thirdly, in soaking the foot night and morning in alum lotions, or brine, or solution of tannin. Thread and cotton coverings for the foot should never be worn. When wool or merino cannot be borne, silk is the only proper substitute.

Until comparatively lately the shoes supplied to the English army were symmetrical, that is to say, there was no difference between right and left. This is now happily corrected owing to the efforts of Dr Parkes and other medical reformers; at present our soldiers are probably better shod than the French with their shoes and gaiters, or the Prussians with their unyielding boots. The best foot covering of all is, I believe, a kind of sandal worn by the Spanish infantry. In the handsome and serviceable costume of the Hungarian army, an excellent laced boot is worn, much like that of our own troops, but somewhat higher, like a shooting boot, without the addition of a leather legging. The importance of anatomical knowledge in army clothing is conspicuous in this instance; but in civil life, apart from artistic considerations, the misery and ill-temper produced by ill-fitting shoes renders the subject one of serious importance.

Leucoplacia buccalis.—Ichthyosis linguæ.—Closely allied anatomically to corns and callosities, consisting like them in hypertrophy of epithelium, are the milk-white patches or corns upon the prominent parts of the heart, both auricle and ventricle, and the thick gristle-like white fibrous patches on the surface of the spleen, with similar conditions less frequently met with in the pleura and the peritoneum.

Still more closely connected with corns are the white patches (leucoma, leucoplakia, tylosis) upon the mucous membrane of the tongue and inner lining of the cheeks. These patches, which have been described, in addition to the above names, as "psoriasis of the tongue," "ichthyosis lingualis et buccalis," and "keratosis lingual et oris," are of much diagnostic interest. They occur as the result of irritation from a rough tooth. They also are produced or at least aggravated by smoking, not, I believe, by the chemical action of nicotin, but by heat and friction, or the two combined. Very similar patches may be the result of syphilis, but I believe that they may generally be recognised by their not being confined to the mucous membrane but dipping beneath it: moreover, in most cases there is either an ulcer on the patch or more or less contraction around it from previous loss of substance. Beside the diagnosis from syphilis, which is sometimes difficult, these white patches may be the seat of subsequent cancer. They are not its first stage, for they may last many years before malignant action appears, but they are the seat of irritative proliferation of cells, which only needs the determining conditions, whatever they may be, to produce carcinoma.*

WARTS.—*Verrucæ, papillomata.*—These are small cutaneous tumours consisting in overgrowth of the papillæ of the cutis.

A vertical section shows that the horny layer of epidermis is unaffected or is somewhat thinner than usual. The Malpighian layer is sometimes slightly thickened, and in many cases is the seat of more abundant pigment than usual. There is seldom or never any evidence of inflammation, the process is one of hypertrophy and new growth.

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* See the discussion on this affection at the International Medical Congress, 1881 (vol. iii, p. 171), introduced by Dr Schwimmer, of Buda-Pesth.

Warts are very rarely painful, but their removal is desired from their unsightliness and also because of their inconvenience, or sometimes the pain occasioned when they are accidentally pressed upon. They are sometimes single, more often multiple, and in rare instances occur in innumerable multitudes. They appear to be never congenital, but are most common in children and are comparatively rare after early adult life. We can sometimes trace their origin to certain definite sources, usually some form of local irritation.

The most common seat of warts is on the hands, not the palm, but the fingers, the dorsum, and the wrist. They may also occur on the arms, the face, not unfrequently on the scalp, and more rarely on the trunk or lower extremities. They are decidedly rare on the feet but are not uncommon on the penis and vulva, around the anus, and at the orifice of the lips and on the mucous membrane of the mouth. A similar condition occurs also in the œsophagus, especially in certain cases where pressure has produced irritation of the mucous membrane and also where chronic cardiac disease has led to its habitual congestion. Warts are usually of a rounded, hemispherical, or pointed shape, but sometimes are flat at the surface, and by growth or coalescence a large flat warty mass may be formed which is called a condyloma.

Pathologically we may recognise the following varieties of what it is impossible to separate by any sharply defined line as anatomical structures:

1. The innocent and painless warts of youth, chiefly occurring on the hands, easily removed, not recurrent, and if left alone probably disappearing of themselves. When one of these appears others quickly follow, and their prevalence among children of the same age has led to the popular belief that they are contagious. They are almost always found upon the hands.

2. Small multiple warts, usually of a pinkish colour and thus differing from the pale or yellowish tint of those first described. Beside their small size and colour they differ in their often occurring in large numbers so as to simulate papular dermatitis, or, again, if rather large, discrete, and somewhat flat they may simulate molluscum, a variety of which has been named "verrucosum" from this resemblance. These multiple warts I have usually seen covering the arms, but I have also met with them on the neck, face, and forehead. In one case, in a girl of eighteen, they covered the back of both hands, in another of a healthy woman of twenty-eight, they closely resembled lichen planus. In a third, a young man of twenty, they occupied the neck and left side of the nose, where I counted more than three dozen. He had also warts, though less numerous, on both hands and forearms. They all came in six months, starting with one large one on the pomum Adami. This patient had had warts on his thumbs when a boy.

3. Warts of old age, usually single or few in number, large and deeply pigmented. They are apt to occur around the orifices of the body, on the eyelids, the lips, the genitals, and around the anus. Similar papillomata occur on the tongue and mucous membrane of the mouth. They are very liable to degeneration and are often the seat of subsequent epithelial cancer after having existed for months or years without showing the slightest malignancy.

4. *Condylomata and mucous patches.*—The composite warts, known as condylomata, hard condylomata, Spitzcondylom (*C. acutum*), are true papillomata in structure, but are always local, never scattered about as true warts are. They occur most frequently about the anus and genital organs. They are certainly not always syphilitic. They may follow the irritation caused by the discharge of a soft chancre or a gonorrhœa, in the latter being identical in all but size with gonorrhœal warts. I have also seen them as the

result of friction from riding in the cleft of the nates, where there was not the least probability of other than mechanical origin.

On the other hand, the soft condylomata (plaques muqueuses or mucous patches) are believed to be always syphilitic, and they almost alone of secondary lesions have the power of transmitting the virus. They occur on the lips, on the mucous membrane of the tongue, cheeks, palate, and tonsils, occasionally on the eyelid, sometimes on the female mamma, and frequently around the anus and vulva. Here they may grow to great hypertrophic masses, the tertiary syphilis vegetans of authors. Their treatment will be found mentioned in the account of syphilitic affections in the first volume.

Ichthyosis.—This is a very remarkable and in its fully developed form a rare affection. It is an example rather of hypertrophy and malformation than of chronic inflammation. It was classed by Willan among his squamæ, and its name, "the fish skin disease," was given for the same reason. The scales of ichthyosis are, however, very different from the branny desquamation which follows all superficial dermatitis, from the large pearly coherent scales of psoriasis and from the thin squames of pityriasis rubra. In the most marked cases the surface of the skin rather resembles the rough, dark, and scaly surface of the bark of a tree or it may be compared to the rugged hide of an elephant. Sometimes the roughness and horny excrescences are so marked that they rather resemble the prickly skin of certain sharks, so that persons affected with an extreme degree of ichthyosis have been exhibited as "porcupine men."

The disease is congenital, but does not appear until infancy is past, although the mother will generally admit that the infant's skin was from the first more smooth, dry, hard, and shining than that of other children.

Ichthyosis is most marked upon the limbs, but its characteristic feature is that it is practically universal. In a fully developed case no portion of the whole body is absolutely healthy. The parts least affected are the scalp, face, palms, soles, genital organs, and the flexures of the joints, in other words the thinnest portions of the skin. The greatest accumulation of horny epidermis is on the outside of the arms and legs, and especially about the elbows and knees, but the back, the buttocks, and the whole of the trunk are often scarcely less affected. The scales are not large and are more adherent than those of psoriasis, so that considering the thickness and extent of the mass there is less free desquamation than one expects. The surface is dry as well as rough; there is almost complete absence of perspiration; the sebaceous glands, instead of their natural lubricating oil, secrete a thick material (seborrhœa sicca) which helps to form the bulk of the crusts and gives them more power of attracting and retaining dirt. The difficulty of keeping the rough scaly skin clean is extreme, so that children affected with it have a dingy appearance, which in some cases and in the worst parts becomes almost black. The name "ichthyosis nigra" has been very unnecessarily applied to this condition. There is no deposition of pigment, and I have observed that mere friction will sometimes rub off the superficial dirty scales from the most exposed parts and leave a grey abraded surface which is characteristic and hideous enough. The thick dry skin is apt to crack in a somewhat regular square fashion like the skin of certain armadilloes, and these cracks may penetrate to the cutis and become painful bleeding rhagades.

Except for this accident, ichthyosis is completely painless and apparently does not affect the general health. One or two children under my care affected with it have been remarkably plump, rosy, and in other respects well developed and healthy.

As I have above remarked, ichthyosis is a congenital disease or rather malformation, and it is not unfrequently seen in families, as in the famous case of John and Richard Lambert, two brothers who were exhibited as the porcupine men, and whose father is said to have had a similar state of skin.

Histology.—On vertical section the diseased masses are seen to consist of beautifully arranged wavy layers of horny scales exactly like those of the thicker parts of the cuticle. A section of the skin shows that the Malpighian layer of epidermis is proportionately small, and that the ridge-and-furrow cells have more or less completely disappeared; in other words the keratin transformation of epithelium is here more rapid than usual. The cutis is completely unaffected. Contrary to the statements of earlier writers, the independent observations of Fagge and of Esoff first showed that although the papillæ are often elongated, this is really a secondary change,—that they are atrophied and not hypertrophied. The sweat-glands have disappeared or only exist as cysts, and the sebaceous glands are smaller and less numerous than usual. The hair-sacs are thickened by overgrowth of epidermis and the hairs are atrophied, tufted at the root, and easily shed.

Pathology.—If the above account of ichthyosis be correct, there is no need for the distinction which Erasmus Wilson attempted to make between true and false ichthyosis. His false ichthyosis, the ichthyosis sebacea of other authors, is the seborrhœa sicca above described (p. 685). In true ichthyosis there is no doubt a certain amount of sebum, which is mixed with the epidermic masses, and can be extracted by ether in the form of stearin and cholesterin; but this is not the essential part of the disease. In opposition to Wilson, Begbie, and Tilbury Fox, Dr Fagge argued in the 'Guy's Hospital Reports' for 1870, p. 310, for the view which I have above stated.

Nor is there any need to continue the distinctions of Devergie and other writers into Ichthyosis alba, I. brunea vel nigra, and I. hystrix, or Alibert's of "Ichthyose nacrée and I. cornée."

Xerodermia.—We must, however, recognise as true ichthyosis, though of a much milder form, that affection of the skin which was named by Wilson "xeroderma."* This dryness of the skin is characterised by very obvious roughness to be felt rather than seen, chiefly affecting the outside of the arms and legs. There is but little desquamation, and the morbid change is so slight that it is difficult to believe it can be essentially the same as that which produced the porcupine men. But of this there is no doubt, for we meet with every gradation between the two conditions. The *ichthyosis hystrix* of Tilesius is extremely rare, and even the slighter forms of xerodermia are more extensive, obstinate, and clinically important, when carefully watched, than they at first sight appear. At the same time we may admit roughly two groups of the affection, the more severe, which corresponds with the classical description of ichthyosis, and the milder forms, for which the term xerodermia may be conveniently used. Both alike are congenital malformations, both have the same distribution and probably the same anatomy. I have never seen a section (or even a drawing of the microscopic appearances) of the skin in the milder form of the disease. Each case has its own characters from an early period, and when once established in the second or third year of life, does not apparently become very much worse.

The chief importance of this remarkable disease, even in its mildest form and quite apart from the hideous deformity of the worst kinds, is that the dry, harsh, unlubricated skin is extremely disposed to superficial dermatitis; or, as it is usually put, ichthyosis and xerodermia are often complicated by eczema.

Treatment.—The first indication is to cure the inflamed, red or weeping patches and the deep painful fissures by the same methods which have been above described for the treatment of eczema rubrum, madidans, and rimosum. The second indication is to supply the deficient natural lubricant of the skin by oils or ointment; suppleness is thus restored, the characteristic dryness is removed, and the liability to dermatitis reduced to normal limits. In the more severe cases, however, it is necessary before this can be done to remove the products of disease; and for this purpose warm baths, alkaline baths, friction with soap and water, and above all with soft soap, are the measures which are necessary. The only caution is not to be too vigorous in softening and removing the diseased epidermis, until local inflammation has been relieved. From time to time the process of cure may have to be interrupted and the tender skin soothed by zinc or lead ointments or olive oil. Dr Fagge, as also Dr Liveing, recommended glycerine of starch, but I have found that oil is far more soothing than glycerine in any form.

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* In this, as in other similar compounds, the name of the disease should always be spelt with *i*. So sclerodermia, anæmia, and anuria.

It is astonishing what excellent results may be obtained, even in the worst cases of ichthyosis, when so treated with perseverance and an intelligent appreciation of the object in view. Within a few weeks children, whose portraits would almost go side by side with that of the porcupine men, present an appearance which it requires the scrutiny of an experienced eye to recognise as more than "a little roughness of the skin."

The disease, however, is relieved, not cured. As soon as the patient is neglected it returns as before, and he can only maintain his skin in bearable condition by constant attention to cleanliness, by frequent warm baths, and continual inunction. I have not found internal treatment necessary, but in the article above referred to Dr Fagge recommends antimonial wine and many physicians administer cod-liver oil.

Cornu cutanum (*ichthyosis cornea* of Willan and Bateman*) is the name applied to those remarkable cases of horny growths which have been frequently regarded as "freaks of nature."

The growth can always be readily removed, and shows no tendency to return; although, as Bateman remarks, if merely sawn or broken off, they invariably sprout again, like hair or nails.

Five remarkable cases of cutaneous horns, one on the neck and the other on the hand, were modelled by the late Mr Towne for the Guy's Hospital Museum (Nos. 333 to 339).

The term *ichthyosis congenita* has been applied to a rare and remarkable form of disease described by Lebert in 1864 as *keratosis diffusa intrauterina.* It affects the whole of the skin with thickening of the epidermis, which is too small for the body, so that the child is literally hide-bound. Numerous and deep fissures result, and the appearance which ensues has been described as the "harlequin fœtus."

The horny layer is enormously increased, the papillæ and the rest of the cutis unaffected, the sebaceous glands atrophied, and the ducts of the sweat-glands enormously stretched.

Cases of this curious and very rare affection have been described by several authors. The best account of it is that given by Hans von Hebra in his 'Krankheiten und Veränderungen der Haut,' p. 348.

Sclerodermia.—This is a somewhat rare but interesting disease of the skin, the pathology of which is too little known for us to class it definitely among chronic inflammations, or atrophies, or new formations. I place it here for convenience.

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* Bateman objects to calling them horns on the ground that they have no connection with the bones or other part beneath, and are of purely cuticular growth. But this is really the only ground on which we call them true horns and not exostoses or antlers. What he meant was that they have no bony core as the horns of ruminants; but they are exactly identical in structure with that of the rhinoceros.

It was first accurately described by Thirial. One of the best contributions of the lamented author of the present work to dermatology was his masterly account of this disease in the 'Guy's Hospital Reports' for 1867, in which he conclusively proved the essential identity of the diffuse sclerodermia of authors with the circumscribed sclerodermia which was also known as *Addison's keloid*, and is synonymous with many cases described as *morphœa* by older writers. See also his second paper (ibid., vol. xv, p. 297).

Course.—The disease begins very gradually in a hardening of the deeper layers of the skin. The epidermis is unaffected, the surface smooth, and the colour unaltered; but the patient finds that the affected spot is stiff, and on feeling it, a more or less marked induration is recognised, the skin cannot be pinched up into folds as in health, and instead of the natural elastic softness of the integument, a characteristic hardness appears. In the circumscribed form the edges are well defined, so that it feels as if a disc of hard, smooth leather were let into the skin. In the diffused form the stiffness and induration become gradually less and less until they are lost in the natural softness of the skin, but even then it is, I think, usual to find some directions in which the sclerosed patch has a more definite edge. Sooner or later the local appearances become more marked, the affected skin becomes white, or assumes a sallow yellow tint, or becomes pigmented with a pale yellowish brown, which is usually most marked towards the borders and is never uniformly diffused over the entire patch. Quite early a slight rosy circle may be observed around the patch, occasionally forming a distinct ring in the circumscribed form or a more ill-defined and irregular blush in the diffused form (*scleriasis*). The smooth white patch, with its colour heightened by the pink margin, has been often compared to an ivory disc.

The patch of morphœa may go on increasing until a disc several inches in diameter is formed, or it may lose its distinctive characters and pass into the diffused variety. Diffused scleriasis usually has its own characters from the beginning, and extends with no definite margin until it involves a considerable part of one limb, or one side of the neck or half the trunk. After a time contraction begins to appear and scar-like bands vary the surface of the disease. This, together with increasing pigmentation, gives some resemblance to the contracted cicatrices from a scald or burn, and explains Addison's application of the term keloid to the affection.

Locality.—A patch of morphœa most often develops on the trunk, particularly on the skin of the female mamma, where such parchment-like *plaques*, or ivory indurations, like the skin frozen by an ether spray, have been sometimes called "vitiligo." Diffused sclerodermia may be seen on the scalp, the forehead, the chin, or other parts of the face; and the expressionless, mask-like aspect it gives to the features is very striking, particularly since the immobility is not uniform but affects one side or certain features only. Scleriasis is also frequent in the arms, hands, and fingers, which become contracted and useless, and on the side of the neck, where a distortion may be produced which resembles torticollis; or it may invade extensive regions of the trunk or lower extremities. It is quite unsymmetrical.

Cases have been described in which it or a similar affection involves the mucous membrane of the mouth.

Symptoms.—Sometimes patients complain of a good deal of pain as well as stiffness in the affected parts, but this is often completely absent. There is no itching and, as a rule, no accompanying inflammation. Sometimes, however, deep circumscribed and very intractable ulcers form on the sclerotic patches, as in a remarkable case brought by Mr. Baker before the Pathological Society (vol. xxxii, p. 261). There is, I believe, no true anæsthesia—at least, as a rule; nor is there hyperæsthesia, but patients may complain that they do not feel as distinctly as on the normal skin.

Histology.—There is no evidence of any true inflammatory process in this singular disease, nor does there seem to be anything which can fairly be called a new growth; the epidermis is unaffected, the papillæ atrophied only in the later stages of the affection, the hair-sacs, sebaceous and sweat glands normal, as also are the unstriped muscles of the cutis. The seat is primarily in the deeper layer of the cutis and the subcutaneous tissue. Here the fibrous bundles become thicker and the fat between their meshes is absorbed, while increased pigment is gradually deposited both in the papillæ and in the cutis. No cell proliferation is to be seen, according to the careful observations of Chiari, in the 'Vierteljahresschrift f. Derm. u. Syph.,' 1868. This process of mingled hypertrophy and atrophy leads to the characteristic results, both of the earlier and later stages of the disease. In the earlier stage, by compressing the blood-vessels, the peculiar pallor is produced; and by the increase of fibrous tissue and disappearance of fat, the scleriasis of the later stages. Beside the pigmentation, the affected parts sink below the level of the healthy skin instead of being, as at first, on the same level, and the contraction leads to the cicatrix-like bands which crumple the fingers or deform the face or breast.*

Prognosis.—Dr Fagge made the remarkable discovery that sclerodermia, both in its circumscribed and diffused forms, is liable to spontaneous involution. He tracked one of the most marked cases described by Addison, and found that the patient's skin had recovered its normal condition. The same thing has been repeatedly observed since, although it is too much to say that complete recovery is an invariable or even a frequent result. The disease, at all events, shows no tendency to develop into any active or malignant form, and beyond the disfigurement and disablement due to contractions and the occasional pain, the most serious result is the rare one of ulceration, as above noticed. It is more common in women than in men.

Efficient *treatment* is completely unknown. I have tried emollient oils, warm douches and gentle repeated manipulation with some apparent benefit. Electricity has also been employed, either in the form of continuous galvanism to the affected patches, or by interrupted galvanism to the neck in the somewhat vague hope of stimulating the cervical sympathetic, and the equally vague expectation that occasional stimulation of the cervical sympathetic would have any effect upon the disease.

Sclerema neonatorum.—This affection, sometimes called Thirial's disease, is best named as he called it, *sclerème*, in distinction from the sclerodermia or scleriasis just described. Scleroma is a useless variation. It is the condition which is known as "hide-bound" in newborn children, affecting the whole of the surface, and characterised not only by hardness, want of elasticity, and pallor, but also by œdema. The temperature is lowered and the child generally dies within a fortnight.

Linear atrophy.—Somewhat resembling sclerodermia in appearance, and perhaps also in pathology, is a curious affection of the skin, which takes the form of long streaks, generally broader in the middle than at the ends, or less frequently, of round, more or less regular patches: in both cases it appears like a scar, for there is loss of pigment and atrophy of the cutis vera. It was first described by Dr Wilks† as an idiopathic affection which exactly resembles the cicatricial marks caused by overstretching of the skin and rupture of its deeper layers—well known under the name of lineæ gravidarum as a result of abdominal distension from pregnancy, but also seen in ascites or whenever the abdominal skin is similarly stretched, and over joints which have enlarged and stretched the skin. The spots are palpably depressed below the level of the healthy surface, and on a microscopical section, which was carried out by Kaposi at Vienna, the papillæ were found atrophied or vanished, the epidermis in both its layers thinned, and the subcutaneous tissue and glands atrophied. This curious affection, which may be quite idiopathic, has been seen upon the hips, the leg, the knee, the ankle, and the hand. In the early stage the marks are somewhat pink, but there are no signs of inflammation, no pain, or any other symptoms. In a case described by Dr Liveing the maculæ were at first slightly red and raised above the skin; they occupied the upper part of the sternum and neck, and after passing into the atrophic stage above described, ultimately underwent gradual involution.

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* I am indebted to Dr Van Harlinger, of Philadelphia, for a valuable paper on "Sclerodermia," with full list of references, reprinted from the 'American Journ. of Syph. and Derm.,' October, 1873. See also an account of the Histology of a Morphœa Patch, by Dr Crocker, 'Path. Trans.,' 1880.

† 'Guy's Hosp. Reports,' 3rd series, vol. vii, p. 298.

There are some good models of these striæ atrophicæ in the Guy's Hospital Museum, Nos. 340—347.

Elephantiasis.—*Elephantiasis Arabum; elephas; bucnemia* or *bouknemia; dal fyl—pachydermia.*—These names have been given to a curious form of hypertrophy of the skin almost peculiar to tropical climates. It is not necessary to enter upon the tangled labyrinth in which this and so many other names of cutaneous diseases are involved. It will suffice to say that the word elephantiasis was used by Aretæus and by Celsus for the very different disease known to the Greeks as lepra and to English readers as leprosy. They used it because of the magnitude and monstrosity. Unfortunately the comparison was supposed to be between the appearance of the disease and that of an elephant's hide, and since the legs affected with pachydermia have some resemblance to the swollen and shapeless limbs of an elephant, the two diseases and their names were long almost hopelessly confused.

The most important pathological fact about elephantiasis, using the term as applied by the Arabian translators of the Greek authors, is that it is hypertrophy dependent upon recurrent deep dermatitis. All observers in countries where the disease is endemic agree that it begins and is accompanied by recurrent attacks of what has been called erysipelas, each attack leaving the tissues more thickened and infiltrated. Inflammatory œdema of the skin and subcutaneous tissue is the characteristic lesion. This gradually becomes œdema durum and no longer yields to pressure, the infiltrated tissues undergo hypertrophy, and masses of fibrous tissue are thus produced, which may be described as a diffused new growth. The skin itself appears at first to be unaffected, at least in its papillary and epidermic layers; but after a time it also hypertrophies, the papillæ becoming enlarged and the surface coarse, thick, scaly, and pigmented.

Histology.—On section, the hypertrophy of the deep layer of cutis, and the massive fibrous bands of white and elastic tissue, with œdematous connective and adipose tissue, are very characteristic; the lymph-spaces of the cutis are enlarged, and the lymphatic vessels are frequently found dilated and varicose. Occasionally an ulcer will accidentally open one of these enlarged lymphatics; and a discharge of normal lymph, more or less milky if it has passed through several lymph-glands, is poured out.

The disease never goes below the integument, it does not spread to the deeper fasciæ or bones, and it never affects internal organs or leads to any but local results.

Such a condition is occasionally seen as the result of long-continued inflammatory dropsy of one limb. I figured such an example in a case of old dermatitis of originally syphilitic origin in the Guy's Hospital 'Reports' for 1877, plate ii. I have also seen a similar result in cases of enormous obesity and general hypertrophy of fat and subcutaneous tissues. Or, again, it may be the result of local pressure upon the veins and lymphatics, as by enlarged inguinal glands or other tumours. But in many hot countries, particularly the West India Islands, where the disease is known as the "Barbadoes leg," in Cape Colony, Egypt, South America, in China and Japan, and in the Pacific Islands—elephantiasis is idiopathic and endemic. My friend, Dr Turner, of Samoa, has sent me numerous figures of this disease, which presents exactly the same features there as in the other races and climates where it is found.

The *distribution* of elephantiasis is almost limited to the legs and scrotum. Sometimes only one foot is affected, sometimes the thigh remains free. One leg may entirely escape while the other forms a huge tumour, and the scrotum may be diseased independently or along with the legs. The size of these scrotal tumours is sometimes enormous, the mass reaches to the ground, the penis is completely lost within it and the whole weight exceeds that of the rest of the patient. As above stated, the organs involved in this monstrous tumour are, when dissected out, found perfectly normal, except that the tunica vaginalis is often the seat of hydrocele. Ulceration of the unwieldy mass of flesh often occurs, and the pain and discharge of the ulcers may produce a certain amount of cachexia.

The *cause* of the disease was until lately perfectly unknown, but owing to the remarkable discoveries made by Dr Lewis, Dr Manson, and other observers, it is now known that a certain proportion of cases of elephantiasis, particularly when it affects the scrotum (known as "lymph scrotum"), coincide with chyluria and the presence of a parasitic worm in the blood. Of this an account will be found at p. 498 of this volume.

It is supposed that the lymph-channels are mechanically blocked by the parasites, which leads to the œdema and inflammation on the one hand, and when rupture into the urinary tract occurs, to chyluria on the other. But there is no doubt that many cases of elephantiasis have been observed in which no filariæ could be detected in the blood. See a case with discharge of milky lymph recorded by Mr Wagstaffe ('Path. Trans.,' 1875, p. 215), and in the same volume, one with great lymphatic dilatation figured by Mr Stewart, as well as a third case of ordinary pachydermia with histological figures by Mr Butlin.

The *treatment* of this disease is purely surgical. There appears to be little or no power of restraining its course until the tumour is sufficiently large to be removed. From the famous cases of Clot Bey in Egypt to those of Dr Turner and other medical missionaries, the removal of these frightful masses of flesh has been one of the most brilliant benefits conferred upon inferior races by European surgery.

Xanthelasma, which as a chronic deep dermatitis comparable to atheromatous inflammation of arteries might find its place in this chapter, has been already dealt with in connection with jaundice (p. 280).

Of the other deep chronic inflammations of the skin, syphilitic affections have been described in the first volume (p. 116); lupus and leprosy both partake of the character of new growths, and will be treated separately in the two following chapters.

LUPUS

Definition, history, and nomenclature—Anatomy and course—Locality—Symptoms—Age—Geographical distribution—Histology—Diagnosis from cancer, rodent ulcer, and syphilis—Pathology—Relation to Tubercle—The "scrofulides"—Prognosis—Treatment.

Of all diseases which affect the skin alone, lupus is the most destructive. Unlike syphilis, leprosy, and malignant growths, it is a purely cutaneous disease. It affects the deep layer of the cutis, and the epidermis and papillæ are only involved subsequently. It also spreads to the subcutaneous tissue and occasionally to adjacent mucous membranes, but rarely or never to the cartilages or fascia propria beneath the skin, and certainly never attacks muscles, bones, or other deeper structures. Lupus is at once a chronic deep dermatitis of a special kind and a new growth in the modern sense of the word. Virchow included it with tubercle, leprosy, and syphilis among the Granulomata or new growths which consist of the same corpuscular elements which form the granulations of a healing ulcer. By the French writers it is almost universally assumed to be tubercular in nature, and forms the type of a supposed natural family of Scrofulides. In earlier times lupus was confounded with ordinary chronic ulcers, with cancer, with leprosy, and most of all with the later forms of syphilis. The lupus exedens of older writers was in most cases tertiary syphilis, as is seen in the woodcut given as a type of the disease in Druitt's 'Vade Mecum.' *Noli me tangere, tentigo prava,* and *herpes exedens* were old synonyms of lupus. The word "lupus," which has been traced back by Virchow to the school of Salerno in the thirteenth century ('Archiv,' vol. xxxii, 1865), expresses the destructive ravages of the disease. It was first carefully defined and described by Willan, and figured by Bateman in his sixty-seventh plate. He places it among tubercula and notices its characteristic proclivity for the face. The distinctions subsequently introduced between lupus exedens and non exedens (Rayer), the lupus "qui détruit en surface, en profondeur, et avec hypertrophie" (Cazenave and Schedel), lupus serpiginosus, syphiliticus and vulgaris—with many others—appear to me to be unnecessary. Only one aberrant form, or rather perhaps one closely allied disease, lupus erythematosus, need be separately named.

Anatomy.—As above stated, lupus begins by the formation of minute nodules of granulation-tissue in the deeper layer of the cutis. These can be felt like shot in the skin, although without any hardness, and show as reddish spots ("macular" stage). When exposed, they are found to be vascular; and when several are seen together, a yellowish tint is sometimes observable, which has been compared to that of apple-jelly.

Histologically, they consist of small nucleated exudation-cells with very scanty stroma. As the disease goes on, these granulation-nodules unite, and undergo changes in two directions. The intercellular substances may become a stroma of delicate connective tissue. This may increase and acquire firmness until it becomes connective tissue with spindle-shaped corpuscles; and finally may form a firm, fibrous, contracted, or œdematous tissue, which resembles an atrophic or hypertrophic cicatrix. More frequently, however, either universally or with only a certain amount of the fibrous transformation just described, the new-formed lupus tissue breaks down, the nodules become confluent, the cells undergo fatty degeneration, caseous softening destroys the new growth, the epidermis gives way, and an ulcer results. The floor of this ulcer is formed by lupous nodules which can be distinguished by the naked eye from the healthy granulations of a healing sore. The edges are somewhat raised and can generally be felt to consist of tubercles which have not yet softened. The pus secreted is usually thin and scanty. While fresh deposition of nodules and fresh softening and ulceration ensue, there is usually some effort at repair by the fibrous transformation above described (Auspitz and Thoma).

The whole process is strikingly similar to that which occurs in the lungs during the course of phthisis. There also we have minute nodules of granulation tissue, which have been described both as new growths and as inflammatory. There also the nodules undergo caseous degeneration, softening, and ulceration; the ulceration spreads, with chronic inflammation and continual deposit of fresh "tubercles." There also the caseous and ulcerative process is rarely unaccompanied by some amount of fibrous transformation, which in favourable cases leads to the involution of the disease and the formation of a cicatrix. The bearing of this resemblance on the theory of lupus we shall presently refer to.

To complete the account of its anatomy we must add that the skin around, though red and slightly swollen, is not the seat of active inflammation, the redness is of a venous tint, and the surface does not feel hot.

The process above described is extremely slow. I have watched lupus for more than a year before it has ulcerated. It usually begins at a single spot and spreads irregularly therefrom, sometimes in a serpiginous form and comparatively swiftly, more often with an irregular rounded shape. It is rare for two independent foci of lupus to be seen, but this may sometimes occur. It is decidedly unsymmetrical, unless by accident when it begins in the median line.

Locality.—Lupus by preference attacks the face, particularly the alæ of the nose, the edges of the lips, the cheeks, the eyelids, and the conjunctiva. It often also occurs upon the ears and spreads to the neck. It is rarely seen on the scalp and is not common on the trunk and limbs; but there is probably no part of the body on which lupus has not been observed, and although, as just stated, it is rare to see two lupous ulcers at once, it often after appearing and being cured upon the face, reappears in another region.

In Vienna the trunk and the buttocks are said to be more often the seat of lupus than the arms and legs; the hands and feet are almost exempt.

Lupus also affects the mucous membrane of the nose, the lips, the hard and soft palate, and the larynx (*cf.* vol. i, p. 813). I have never seen it affect the tongue or the deeper mucous membranes.*

Symptoms.—As the progress of the disease is slow and its local signs torpid, so its symptoms are but slight. It is astonishing how little pain is felt even when extensive tracts of the skin are deeply ulcerated; as we shall presently see, with regard to pain, the remedy is far worse than the disease. The general health is also unaffected, so that except for the disfigurement and discomfort of having sore places to dress, lupus is one of the most easily borne of all serious and destructive diseases.

Ætiology.—Apart from the question of its relation to tubercle, which will be presently discussed, we have no knowledge whatever of the cause of lupus. It is probably equally common in both sexes. With respect to *age*, it is, as commonly seen, a disease of young adult life. It may however occur in very young children. I have observed it in those not above four or five years old, but it is more common after puberty and usually begins at from fifteen or sixteen to twenty. After thirty it is certainly rare for it to begin, though cases of undoubted lupus may be occasionally observed. Moreover, in young subjects it shows an almost constant tendency to spread, even when repair, as generally happens, is to some extent occurring at the same time; but after thirty lupus tends to undergo involution, and if left to itself will in most cases have ended in cicatrices, disfiguring or disabling but not any longer active.

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* According to Hebra, however, there is no doubt of the cartilages of nose and ears being affected, and even tendons and ligaments of joints. Lupus vaginæ has been recorded.

According to Hebra and his disciples, although lupus is never congenital, it begins before puberty, occasionally in infancy, but usually from the fifth to the ninth or tenth year.

Devergie stated, and I think all physicians will agree with him, that lupus is far more common amongst hospital patients than in private practice. It is also much more common in Vienna than in London. When attending the late Professor Hebra's clinique, I sometimes saw more lupus in a week than one sees in London or Paris in six months. He had no doubt a great reputation for curing it, and the large accommodation at the Allgemeines Krankenhaus allowed of making almost all cases in-patients, but with every allowance for such accidental causes, there is no doubt, from the testimony of others as well as from my own observation, of the frequency of the disease in Austria. Moreover, living as I did in Professor Hebra's house, I had opportunity of observing that in his private practice lupus was almost as common as in the hospital.

Lupus is not hereditary.

Histology.—According to Auspitz, the sebaceous glands are destroyed, the sweat-glands are unchanged, the hair-follicles disappear or are transformed into cysts. Rindfleisch asserts that lupus begins in the sebaceous glands, and this not of the erythematous or so-called sebaceous form, but of lupus vulgaris. He even calls it an adenoma, but this I think there is no doubt is a mistake. Any inflammatory disease will show most exudation in the more vascular parts of an organ, and the most vascular parts of the skin are the papillæ and the sebaceous glands, but the glands themselves are not involved except as a secondary result in the disease. Neither Neumann nor subsequent pathologists agree with the statements of Rindfleisch.

Giant-cells are also observed (Friedländer in 'Virchow's Archiv,' 1874; and Thin, 'Med. Chir. Trans.,' vol. lxii).

Diagnosis.—The fact of lupus being a deep inflammation of the skin and leaving scars, at once distinguishes it from eczema and all the superficial forms of dermatitis enumerated in the earlier chapters of this section; nor is there much practical difficulty in distinguishing it from varicose and other traumatic or accidental ulcers. The real points of diagnosis arise between lupus, syphilis, rodent ulcer, and cancer of the skin.

Lupus is distinguished from cancer by the absence of pain, by its slow progress, by its beginning early in life, by the presence of granulations, the absence of hæmorrhage, and the tuberculated, but not uniformly and densely infiltrated, edge. Invasion of deeper structures and secondary enlargement of the corresponding lymph-glands decides the case to be cancerous, but our object, of course, is to make diagnosis long before this point has been reached.

Rodent ulcer is covered by an adherent reddish-brown scab which when present is characteristic, but it may often have been removed by accident, by poulticing, or by other remedies before the lesion is seen. The edges are neither thick, hard, and infiltrated like those of epithelial cancer, nor do they contain little tubercles as in lupus. Granulations are absent, the ulcer being of the kind known as "indolent," while that of lupus is what is called "weak." Like lupus, the face is its favourite seat, but it is the neighbourhood of the eyes rather than the cheeks and nose which it affects; it is always single and never extends so widely as lupus; it makes no attempt at spontaneous cicatrisation; lastly, it only occurs in those who are past middle life.

Syphilitic ulcers have an undermined, not an infiltrated or tubercular edge; the colour of the surrounding skin is brownish or yellowish, whereas that of lupus is of a more venous purplish-red. In both there may be considerable crusts, forming what is described as rupia and ecthyma in the one case and as lupus pustulosus et crustosus in the other, but when these are removed the more characteristic ulcerated surface beneath will be seen. The scars which result when a syphilitic ulcer is healed, often resemble those of lupus, but they are less apt to be hypertrophied, they are much more pigmented, and seldom present the pink aspect and enlarged veins which are often seen after lupus is healed.

Moreover, tertiary syphilitic ulcers, begin as a rule in the formation of a gumma in or under the skin, deeper than the nodules of lupus and less early affecting the epithelium. In the nose this distinction is most applicable,—lupus begins at the edge of the nostril and slowly creeps on, only affecting the cartilages (if it does at all) in its later stages; whereas syphilis begins in the perichondrium or periosteum and it has already destroyed much underlying tissue before the ulcer on the skin appears. The nose which has lost its tip or alæ has usually been affected by lupus; that which has lost its bridge by syphilis. Extensive disease of the skin, with the cartilages and septum intact, is most likely lupus; a small ulcer, with a deep foul cavity beneath it, and exposed bone and cartilage, is almost certainly syphilis. All the frightful cases of destruction of the greater part of the face and opening of the orbit, pharynx, and posterior nares, which figured in museums and plates as lupus exedens, were tertiary syphilis, neglected or ill-treated, which the better diagnosis and improved therapeutics of modern times have happily banished from civilized countries.

Apart from the local characters, diagnosis between syphilis and lupus will be much helped by syphilitic ulcers being frequently multiple, lupus being very rarely so; by secondary implication of lymph-glands being common, with characteristic induration, in syphilis, rare and only as the accidental consequence of temporary inflammation, soft, and painful in lupus. The syphilitic ulcers are usually accompanied by other cutaneous lesions; the lupous ulcer has no complication. Syphilis begins after puberty, often long after; lupus before puberty or shortly after. Lastly, lupus is a disease of the skin and nothing else, whereas syphilitic gummata and ulcers will be generally accompanied by other signs of the disease in the bones, glands, tongue, or viscera.

The cutaneous lesions of congenital syphilis do not, so far as I am aware, simulate lupus; the early coppery rashes have no resemblance to it and the later gummatous ulcers are exactly like those of tertiary acquired syphilis.

Pathology.—Although it is scarcely possible to confound lupus with the ordinary lesions of congenital syphilis, it has been stated by respectable authorities that lupus is the result of inherited syphilis which does not show itself in the ordinary form. Hebra himself was led to this opinion by a striking case in his own practice, where a syphilitic father had born to him —first, a stillborn child; secondly, one which died in a few months, with the ordinary marks of inherited syphilis; the third survived, after suffering from congenital syphilis of the skin and bones; and the last having been born apparently healthy, remained so for some years, but became before puberty the subject of typical lupus. The case is no doubt striking, and is supposed to show that lupus is the feeblest and most diluted effect of transmitted syphilitic virus; but I cannot see more than a coincidence. No one pretends that a child with a syphilitic father or brothers is thereby protected from becoming the subject of lupus, and occasionally the two diseases must occur in the same family or the same individual. Persons affected with lupus may acquire syphilis, and persons who inherit syphilis may be attacked by lupus. Owing to Mr Hutchinson's classical observations (which on the Continent have scarcely received the credit due to so brilliant and important a step in practical medicine) we can now recognise congenital syphilis not only in infants but in later life. Such persons are not more liable to lupus than others. In fact I do not remember seeing a case of lupus in a patient with Hutchinson's signs; and, on the other hand, one ought to be very sceptical in admitting that congenital syphilis is present when it shows itself by none of its certain and unequivocal characters.

The assumption that there is such a thing as "syphilitic lupus," a kind of hybrid between two diatheses, is to my mind unjustified; and, like similar diagnoses of rheumatic gout or hybrids of scarlatina and measles, is practically as mischievous as intellectually it is unworthy. At the same time I do not deny that the diagnosis between syphilis and lupus is often difficult, and I have more than once mistaken the one for the other.

Is lupus, as the French school assert, a "scrofulide?" This word was invented by Bazin and Hardy in imitation of the Syphilides of Biett and Alibert. Bazin includes among "Scrofulides bénignes," chilblains, erythema, strophulus, prurigo, lichen, eczema, impetigo, and some forms of acne. These are justly excluded by the sounder judgment of M. Hardy, who precisely defines his scrofulides as depending exclusively upon scrofula as the syphilides do upon syphilis, never developing without it, and diagnostic of its presence. He would place under this definition lupus, which Biett had, with his usual good sense, separated from all other diseases without inventing an ordinal name for it, and which Alibert had put with eczema and psoriasis among the dartres. He includes under lupus the four species described by Cazenave, erythematous, tubercular, ulcerous, and hypertrophic. He rightly regards the two latter distinctions as unnecessary. Bazin's division of the "Scrofulides malignes" is into erythematous, tubercular, and scrofulide crustacée ulcereuse; and Hardy addresses the same "reproach" to the third of Bazin's as to the last two of Cazenave's species. He himself describes five varieties of scrofulides:

1. *Scr. erythemateuse.*—This corresponds to erythematous lupus, an undoubtedly distinct form, which will be described below.

2. *Scr. cornée et acnéique.*—This is not what other French writers describe as acné cornée, a curious affection of the sebaceous glands unaccompanied with inflammation ("ichthyosis follicularis") very rare, and in the cases I have seen without the slightest claim to the epithet scrofulous (p. 685). Groups of comedones are described by Hardy as placed on a purplish-red patch, and as followed by depressed cicatrices unpreceded by ulceration. It appears upon the face, has a very slow course, and I presume occurs only in persons who for some other reason are entitled to the epithet scrofulous.

I think that in London, and probably in Vienna, we should call this affection lupus erythematosus or lupus sebaceus indifferently or conjointly.

3. *Scr. pustuleuse.*—This is the most frequent variety; it begins either by a number of pin's-head pustules grouped on a small red patch, lasting from a week to a fortnight and leaving a yellow scab, or else with a large pustule, like that of ecthyma, which when ruptured gives place to a dark prominent crust that the older dermatologists would have named rupia. The part usually affected is the nose, the course is very slow and unaccompanied by itching or pain, but the most characteristic point is that when the crusts, which are very adherent, are removed, ulceration is found beneath.

This ulceration is not deep, the surface is pale and sometimes presents little hard, dry, rough, warty nodules, which led Hardy originally to describe the variety as "scrofulide verruqueuse."

This form would by German, English, and American dermatologists be recognised as typical lupus (*Impetigo rodens*), which, as above described, frequently begins in pustules and is accompanied by large scabs. The slow course of the disease makes the subsequent ulcerated stage much more familiar, but even if watched from the beginning I think that, at least as we see lupus in London, cases with pustules and large prominent crusts would be in minority, not less perhaps than a third or a fourth of the whole.

4. *Scr. tuberculeuse* is divided again into a superficial and a deep variety, and the former distinguished as sometimes disseminated over various parts of the body and sometimes localised, sometimes inconspicuous and ending in a slight atrophic scar, sometimes hypertrophied, especially when it affects the genital organs. M. Hardy speaks of these deeper tuberculous scrofulides as producing "ces vastes descriptions, ces plaies épouvantables et hideuses qu'on ne rencontre que trop souvent à la face," and as occasionally proving fatal with profuse suppuration, cachexia, and hectic. This form, according to the eminent author quoted, produces enormous cicatrices on the eyelids, the lips, the neck, the ears, the nostrils, like those produced by severe burns.

This is obviously lupus exedens in its severest and most destructive form, not, I venture to think, differing from the slighter forms accompanied with true ulceration, except in degree. Moreover, even when untreated, its ravages, however hideous, are to my mind more remarkable from their contrast with the deeper destruction of syphilis and cancer than from their extent and severity considered as a disease of the skin alone.

5. *Scr. phlegmoneuse.*—This is a superficial ulcer which begins as a phlegmon as big as an almond or a nut; this gradually softens, fluctuates, acquires a purplish-red colour, and at last discharges a little thin pus; a scab forms, and this process may be repeated and become chronic until a large surface becomes ulcerated. The disease appears chiefly on the face, but also on the trunk and limbs. It always leaves a scar, at first violet-coloured, afterwards pale, irregular, and reticulated.

This somewhat rare variety will be recognised as what older surgeons, and especially I remember the late Mr. Hilton, used to describe as "scrofulous ulcer." The variety no doubt deserves mention, but regarded histologically, or from the point of view of pathology or of treatment, it also may, I think, be fairly included as lupus. When, however, it occurs on other parts of the face, the primary abscess is often due to suppuration of a true scrofulous or tubercular lymph-gland, of which there are not a few very superficial and too small to be recognised in anatomy, which become apparent when enlarged by the hypertrophy of Hodgkin's disease or the inflammation which results from caseous enlargement or any other cause.

A review of these varieties of the scrofulides defined by the most experienced and the most rational of the successors of Biett, and described in his admirable 'Leçons' with the clinical acumen and skill characteristic of Professor Hardy, shows, I think, that the only diseases of the skin which have any title to be called scrofulous are those which Willan and Bateman, with their successors in England, and Hebra, with his disciples in Germamy, would agree in calling lupus. It is remarkable that the rare papular affection of the skin described by Hebra as lichen scrofulosorum (p. 640), and also the dry, harsh, unoiled conditions called xerodermia (p. 686), and pityriasis tabescentium, are not included in the above account of scrofulides.

What ground then is there for ascribing lupus to Scrofula? This raises the question of the meaning which we attach to that much-abused word.

Scrofula originally denoted a swollen neck, which in some children makes the head pass into the shoulders with scarcely any constriction, as it does in a pig (*scrofa*). It is found that this usually depends upon a chronic caseous enlargement with characteristic suppuration and subsequent cicatrices of the cervical lymph-glands. (The word *struma* also meant a swollen neck, and while in England used as a more or less vague synonym of scrofula, which had better be discarded, in Germany it is applied to another chronic swollen neck, namely, bronchocele or goitre.) Scrofula, therefore, may be defined as chronic caseous enlargement, with characteristic suppuration and subsequent scarring, of the cervical lymph-glands.

The mere form of degeneration is, as Virchow long ago pointed out, not characteristic; for it occurs in traumatic abscess and in atheroma, in the midst of tumours and even of cancers. Moreover, most cases of caseous disease of the lympharia would, I think, on careful examination be found not to be idiopathic but secondary to mucous or cutaneous irritation. If indurated lympharia are discovered, we at once seek for a primary affection in a chancre, if cancerous in a primary tumour on an epithelial surface, if suppurating in a primary wound or inflammation of the skin or mucous membrane. In the same way caseous lymph-glands can generally be traced to chronic inflammation of the surface from which they receive their lymph. In the neck most frequently it is traceable to the throat with its tonsils and other lymphatic organs, more rarely to the scalp, the teeth, or the ear; bronchial lymph-glands become caseous in consequence of chronic or repeated subacute bronchitis and broncho-pneumonia; mesenteric lympharia in consequence of chronic or subacute enteritis and diarrhœa. These three groups of lympharia in the neck, the thorax, and the abdomen are the principal seats of so-called scrofula, and the reason is probably because the mucous membrane of the fauces, the bronchial tubes, and the small intestine is pre-eminently rich in adenoid or lymphatic tissue. According to the more rational believers in scrofula as a diathesis, disposition, or general pathological tendency, caseous disease of lymph-glands is clinically found connected with caries of the bones, with chronic inflammations of the articular ends or of the synovial membranes in joints and with certain forms of catarrhal ophthalmia. On these points I do not presume to speak with any kind of authority; but I have certainly seen many children with caries of bones or chronic inflammation of several joints who had no affection whatever in their lymphatic organs; and one constantly sees children, and sometimes adults, with caseous inflammation of cervical and other lymph-glands which lasts for years without their bones, their joints, or their conjunctiva ever being affected. In reading the classical accounts of Watson and older writers of the two types of scrofulous children, one was always sceptical as to the same morbid disposition showing itself in two such opposite ways; and since the writings of Sir William Jenner and Mr Hutchinson have taught us better, we must, I think, admit that "pretty scrofula" was in most cases true tuberculosis and "ugly scrofula" inherited syphilis.

This brings us to the question of the relation between scrofula and tubercle, and the relation of lupus to each. After phthisis and scrofulous pneumonia had been assumed by older pathologists to be pathologically identical with scrofulous glands and joints and bones, Virchow introduced the light of critical histology into the confused mass of doctrine on this difficult subject. Regarding miliary tubercle as the type of that condition and as essentially a granuloma or new growth of an adenoid or lymphatic type, he defined the scrofulous diathesis merely as "vulnerability," that is, tendency of the organism to react to slight injuries and inability to recover from them. Subsequently, however, scrofula in any definite anatomical sense has again approached tubercle; for, first, the giant-cells of Schüppel which were imagined to be characteristic of tubercle were found also in scrofulous lymph-glands by Friedländer; and, secondly, the still more famous *bacillus* of Koch has been discovered in these same organs.

The nodules and granules of lupus contain a minute bacillus which in form, size, and reaction to staining agents is to my apprehension undistinguishable from that found in phthisical sputum.

Nevertheless, no physician with clinical experience is prepared to admit that the uniform presence of a bacillus, any more than of a histological element or of a chemical product, can settle the true affinities of morbid processes; which must be judged of ultimately by their natural history and physiology, not by their anatomy, chemistry, or mycology. In this as in other matters, to use Hebra's dictum, where the pathologist and the clinical physician differ, clinical knowledge must be the master: "Wo der Patholog und der Kliniker im Streite sind, muss der Kliniker Meister sein." Admitting, then, that the *bacillus lupi* is constant and is a tubercle bacillus, and that the same organism occurs in tubercle, in scrofulous lymph-glands, and in lupus, do we find clinically that lupus occurs in persons who have definite signs of scrofula or who are subject to tubercular diseases?

To the latter question I think we may at once answer, No. It is extremely rare to see lupus among the countless victims of phthisis, *i.e.* of chronic tubercular inflammation of both lungs, beginning at the apex, travelling down, ulcerating and destroying the tissue, and associated with laryngitis, enteritis, and tubercles in the viscera. Nor, looking at the question from the opposite point of view, have I found among patients with lupus, either in hospital practice in London or in the large numbers I saw under Hebra's own treatment, any considerable number of cases of phthisis; remembering how common that disease is both in England and in Vienna, so that phthisis has been regarded by English writers as the characteristic scourge of this country, and by Austrian writers as so peculiar to Vienna that its prevalence has been supposed to depend upon the geological condition of the soil.

On the other hand, I must admit that one does sometimes meet with caseous glands or scrofulous scars in patients with lupus. I doubt whether the occurrence is more frequent than mere coincidence would explain, when we remember that both lupus and scrofula principally affect children and young adults. This very predilection, however, for a certain period of life may be fairly brought forward as an argument for a relation between the two diseases. A more powerful argument to my mind is the considerable resemblance in the mode of treatment which is found effectual for both.

On the whole, I think we must admit that lupus has a certain pathological relation to caseous or tubercular disease of the cervical lymph-glands, independent of its histology and the presence of a bacillus; and that apart from this relation it has no connection with phthisis or with general tuberculosis. In fact, the clinical relation between phthisis and general tuberculosis on the one hand and tubercular or caseous lymph-glands on the other, is a slight and uncertain one. It is also clear, I think, that with the slight and unimportant exceptions of so-called lichen scrofulosus and pityriasis tabescentium, all the diseases of the skin which have any true connection with scrofula or tubercle may be fairly comprised under the name Lupus. Lastly, notwithstanding these concessions we must maintain that it is not justifiable to forsake the old, well-understood, short, and expressive term of lupus, one merit of which is that it expresses no theory and begs no question. In many cases of lupus, those who believe in tendencies and diatheses may fairly call the patient scrofulous, just as in many cases of eczema they may fairly call him gouty, and in certain cases of erythema may fairly call him rheumatic. I would prefer to say that lupus may be called scrofulous if the patient shows scars which prove that he has had caseous lymph-glands, that eczema may be called gouty when it occurs in a patient who has tophi in his ears or in his joints, and that erythema may be called rheumatic when it occurs in a patient who has suffered or is suffering from rheumatic fever. In the majority of cases of lupus, as I have seen it in Vienna, in London, and also in Paris, there was nothing which an unbiased observer would have called a sign of scrofula excepting the disease of the skin.

Auspitz—who has widely departed from Hebra's simple though unpractical classification of diseases of the skin upon the system of general morbid anatomy laid down by Rokitansky—classes lupus among what he styles "chorio-blastosen" or anomalies of growth of the corium and subcutaneous tissue. He subdivides this group into simple hypertrophic (macrosomia) and paratypical or abnormal growth, which includes the granulomata. Here lupus finds a place side by side with leprosy, scrofuloderma papulosum, or lichen scrofulosum and scrofuloderma pustulosum, or acne cachecticorum, scrofuloderma ulcerosum, or scrofulous ulcers of the skin, tuberculosis cutis as a separate condition, syphilis, and, lastly, rhinoscleoroma. Hans von Hebra adopts this arrangement.

This is practically following Virchow's arrangement of tubercle, lupus, and leprosy among the granulomata of his great group of new growths which are framed on the type of connective tissue.

In Ziemssen's 'Handbook' Neisser places lupus close to tuberculosis of the skin and scrofulodermia, and makes them one division of a group of chronic infectious diseases* of the skin, which includes in addition leprosy, syphilis, glanders, rhino-scleroma and frambœsia. He, however, excludes erythematous lupus from the group. Writing in 1883 he admits that no one has established the constant presence of Koch's bacillus tuberculosis in lupus, while Schüler has found only micrococci.

Kaposi and Baumgarten both oppose the recognition of lupus as a tubercular disease. Auspitz and Frederic Lander, Neumann, and other modern dermatologists in Germany admit it, and even Baumgarten allows a possibility of genetic relation between the two. Erasmus Wilson maintained the relation, and Plumbe spoke of lupus as a strumous affection. Dr Fagge says "it is apt to occur in scrofulous persons," and Dr Liveing that "it belongs rather to the scrofulous diathesis." The late Dr Tilbury Fox "could not subscribe to the view that lupus is an evidence of the strumous diathesis, and was more inclined to regard it as having a predilection for tubercular subjects."

Course and prognosis.—Lupus is one of the most chronic of diseases. It creeps on, usually with an imperfect attempt at healing, sometimes retreating until it almost disappears and then again advancing with a persistence and rapidity foreign to its usual character. In the end, if left to itself, it probably heals, leaving, however, indelible marks of its presence in hideous scars, contracted limbs, distorted features, or obliterated orifices. It is singularly free both from pain and from irritation, it never affects internal organs, whatever its true pathology may be, it does not produce secondary caseous inflammation of the lymph-glands which correspond to the affected skin, and never leads to general tuberculosis of the internal organs. Happily it is amenable to the efficient treatment which has been established within the last twenty or thirty years, so that the prognosis almost entirely depends upon the early recognition of the disease by a skilled practitioner.

Treatment.—Bateman remarks that he knows "no medicine which has been of any essential service in the cure of lupus," and that "it requires the constant assistance of the surgeon." Wilson, in the first edition of his treatise (1842) by a remarkable omission, mentions neither the disease nor the name. But the usual practice of the earlier English dermatologists appears to have been to use arsenic and so-called tonics. It was Hebra who, regarding lupus, like most other diseases of the skin, as a purely local lesion, resolutely attacked the diseased tissue, and by destroying it produced a healthy inflammation which ended in cure. The clear insight and determination—sometimes I fear the unfeeling severity—with which he carried out this method, led to the most remarkable success. Tilbury Fox introduced the Vienna treatment into England, and maintained that the real treatment of lupus consists in destruction of the diseased tissue by caustics. Even Hardy, though he begins with general treatment of lupus as a scrofulide, admits that in certain cases local measures are also necessary, that emollient applications are unimportant, and stimulating lotions seldom useful. He recommends caustic iodine—one part dissolved in thirty of water with the help of three of iodide of potassium. Even this he admits is useless in most cases, and recourse must then be had to stronger caustics, of which he recommends chloride of zinc, potassa fusa, and particularly binoxide of mercury.

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* Infectionskrankheit means strictly a disease derived from infection, not one which is itself infectious; but practically the two characters go together.

Often less severe measures suffice, and Hebra himself accomplished admirable results with the solid lunar caustic. A strong solution of the same silver salt (a drachm to the ounce) may sometimes be substituted with good effect. The acid nitrate of mercury may also be applied, especially to small and comparatively superficial spots. But the most satisfactory method of treating most cases of ulcerative lupus is by the sharp spoon introduced by Volkmann, of Halle.* Chloroform should be given and the whole of the diseased surface scraped away. It is astonishing how boldly a skilful surgeon can use this instrument or an analogous one, employing enough force to remove all the diseased tissue without injuring the more resistant healthy cutis which surrounds it. Indeed Hebra's use of the pointed nitrate of silver pencil almost converted it into a scraping or mechanically destructive as well as a chemically destructive agent. The hæmorrhage produced by these operations is less than would be supposed, and it is almost always necessary to repeat the application of the caustic, sometimes again and again, and one advantage of the scraping is that it is sometimes sufficient after a single sitting and seldom requires more than two or three. The saving of time as well as of pain to the patient is certainly remarkable.

Caustic potash applied as it used to be in stick is not only extremely painful, but even with the greatest care will destroy healthy as well as diseased tissue. Hebra's arsenical paste is less destructive, but causes great inflammation as well as pain, and is every way inferior to scraping in its results.

The pyrogallic acid introduced by Järisch is probably the best local application. It should be used as an ointment of 10 per cent. which is better than solution or plaisters. It causes considerable pain, often as much as lunar caustic.

Another plan of treatment also introduced by Volkmann and carried out by Vidal and Besnier in France, by Mr Squire and Dr Stowers in this country, is scarification or, as the operation is now performed, minute stabs with a lancet or an instrument made for the purpose. This section of immense numbers of blood-vessels produces temporary hæmorrhage, but afterwards obliteration of their channels and anæmia of the lupus spots.

The galvanic cautery, though sometimes applicable and less painful than would have been supposed, has the same drawback as caustic potash and sulphuric acid, that is, it destroys diseased and healthy tissues alike.

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* See his paper on "Lupus and its Treatment," translated for the Sydenham Society in 'German Clinical Lectures,' 1876.

I would impress upon the reader the paramount importance, for the successful treatment of lupus, of recognising its character as a new growth which must be destroyed. So long as any of the granulations remain it is liable to return. Once rooted out, it is rare for this to happen or even for it to appear in another part of the skin. Occasionally the knife may be employed to excise part of the tip of an ear or some other circumscribed piece of skin, but scraping or caustic, or the two combined, are in a great majority of cases as effectual and the results better. Indeed when early and thoroughly treated, lupus becomes a manageable disease, and the cicatrices which result are often surprisingly slight.

In the slighter forms of lupus, and especially in the variety to be described as lupus erythematosus, such vigorous means are generally unnecessary, though wherever ulcers or granulations are seen, their destruction by some means or other is the only thorough method of cure. The milder applications which have been recommended, such as tincture of iodine, iodoform ointment (half a drachm to an ounce), pyrogallic acid ointment (a drachm to the ounce), and strong solution of nitrate of silver, may probably stop the disease at an early stage. They certainly check its progress, and may be usefully employed whenever more decisive treatment is counterindicated or postponed. For erythematous lupus especially, pyrogallic acid or iodoform are excellent applications.

Although local treatment is essential for lupus and is often sufficient without any other methods, many dermatologists strongly recommend the internal administration of cod-liver oil. Even Hebra admits its value and used to apply it locally to the sores as well as internally. I confess that I should never advise a surgeon to trust to this remedy without attempting local measures as well, but wherever swollen glands or phthisical symptoms are present, or even when want of weight and flabby muscles show malnutrition, oleum morrhuæ should undoubtedly be given. In the same way the syrup of phosphate of iron, steel wine, or tincture of steel, are indicated by pallor. I do not think that arsenic is of any service in cases of lupus.

Some of the most scaly forms are said to be cured by arsenic, but as I cannot recognise such transition forms as are called psoriasis-lupus, I should suspect these cases of being really psoriasis and not lupus at all; just as I have above pointed out that serpiginous lupus is often extremely difficult to distinguish from syphilodermia, and owing to this difficulty has sometimes been supposed to be cured by iodide of potassium.

Lupus erythematosus.—This curious affection was first described by Biett and named Érythème centrifuge. It was named by Hebra "Seborrhœa congestiva" (1845), and the same view of its nature has led to the titles Lupus sebaceus, Lupus acnéique (Hardy). It has also been named Scrofulide erythémateuse. It is, however, more generally recognised by Cazenave's name, Lupus erythematosus (1850), which Hebra afterwards adopted.

Its essential nature still admits of doubt. There is no question that the sebaceous glands are much affected by it; it is equally certain that a slow chronic dermatitis, accompanied with a violet or rose-tinted erythematous blush, is always present. But it is seldom that one fails to discover evidence of a destructive process of the papillary layer in more or less well-marked cicatrices, and in many instances of undoubted lupus erythematosus the scars are obvious. It is, therefore, I think, rightly associated with the ordinary disease known as lupus, the two forms being distinguished when necessary as lupus vulgaris, lupus exedens, or lupus exulcerans on the one hand, and lupus erythematosus, or erythematodes, or sebaceus or non-exedens on the other.*

The *locality* of this affection is very characteristic. It almost always occupies the face and usually the bridge of the nose, together with the cheek on each side; for, in contradistinction to ordinary lupus, it is remarkably symmetrical. The figure produced by this distribution has been compared to a butterfly, a bat, or the sphenoid bone, and when once seen is easily recognised. It is also seen on the ears. It is sometimes found on the scalp, and then the hair is destroyed, a sufficient proof that lupus erythematosus is not, as it is classed in Ziemssen's 'Cyclopædia,' a superficial dermatitis. It occasionally appears upon the limbs or trunk, sometimes preserving its symmetry, but sometimes being confined to one arm, and most often to the hand. On the trunk and legs it is certainly rare, but in one case I saw it spread over the shoulders and buttocks.

It is seldom that we see the first beginning of this disease. It shows itself as an erythematous patch, not unlike that left after impetigo or an early stage of tinea circinata. It spreads at the edge (whence Biett's epithet *centrifuge*), which is marked by injection, swelling, and desquamation, while the centre becomes pale, smooth, and slightly depressed. One can also usually see the sebaceous glands enlarged, sometimes prominent, resembling acne punctata, sometimes forming black comedones within the affected surface. It thus spreads until it has obtained the form and dimensions above described as characteristic. Sometimes, however, fresh spots occur at a distance, and this is decidedly more frequent than with ordinary lupus. The dry whitish scales formed chiefly of concrete sebum suggested the epithet herpes cretacé to Devergie and sebaceous to Hebra and others.

On making a microscopic section of the diseased patch, infiltration of the cutis with leucocytes and dilated blood-vessels are obvious; and the congestion and proliferation is most abundant around the sebaceous glands. These cells never become caseous or soften down so as to form the granulations and pus of an ulcer. They gradually become transformed into connective-tissue corpuscles, and as the fibres thus formed take their place the papillæ atrophy and the glands shrink and disappear (Neumann, Geddings, Thin). These histological characters appear to me to show that no sharp line of distinction can be drawn between chronic deep-seated inflammation with hypertrophy and consecutive atrophy on the one hand, and development of the simpler forms of new growth like lupus, tubercle, and syphilis. On the other hand, one can, I think, draw a very marked line between deep inflammations with destruction and atrophy on the one hand, and superficial inflammations which do not destroy the papillæ and are never followed by ulceration or cicatrices on the other.

Lupus erythematosus occurs chiefly in adults, but sometimes in children. Veiel says that most cases occur between twenty and forty, and I certainly agree that the average age of this affection is later than that of ordinary lupus. It is perhaps commoner in women than in men. Its course is extremely slow, and like ordinary lupus it is accompanied by neither pain nor itching. It has no claim to be called scrofulous.

Lupus erythematosus in some cases simulates nævus. See the account of patients under the care of Mr Macarthy and Mr Higgins given by Mr Hutchinson in his 'Lectures on Clinical Surgery,' vol. i, p. 284.

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* In favour of this view see Mr. Hutchinson's 23rd lecture ('On certain Rare Diseases of the Skin'). For the severance of lupus erythematosus from true lupus see Kaposi's and Veiel's papers ('Trans. Intern. Med. Congr.,' vol. iii, pp. 162, 167) with comments by Schwimmer and Thin; also Dr Payne's remarks ('St Thomas's Hosp. Rep.,' xiii).

The *treatment* of erythematous lupus is that of the milder forms of lupus vulgaris. Alternatives in the sense described above (p. 737) of a stimulant kind take the place of destructive applications. Hebra's diachylon ointment or solution of soft soap (spiritus saponis alkalinis) sometimes appear to be sufficient. Iodide of mercury ointment (one to fifteen) was recommended by Cazenave; the pyrogallic ointment above described is also useful, but I think I have found the effects of iodoform the most satisfactory of any. In some cases very mild applications can alone be borne, such as unguentum metallorum, yellow oxide of mercury, unguentum hydrargyri ammoniati. In others again the true nature of the disease is shown by the treatment which dermatologists, whatever name they give it, are led to adopt, scarification, and even scraping or the galvanic cautery.

For the local treatment of erythematous lupus Mr Hutchinson strongly recommends the steady use of a weak solution of coal tar. He prefers the ointment consisting of half a drachm of liquor carbonis detergens to an ounce of petroleum gellatum. The unguentum liq. carbonis deterg. of the Guy's Pharmacopœia is stronger.

Lupus erythematosus disseminatus.—Kaposi has named lupus erythematosus as above described *discoid* in order to distinguish it from a rare and remarkable form of disease which he probably rightly regards as a form of lupus, and which he has named the disseminated or aggregated variety of lupus erythematosus. Here the patches do not grow by the enlargement of the circumference, but entirely by fresh ones appearing. Moreover, the disease is not confined to the face, but is seen upon the trunk, the course is sometimes acute, and the whole character of the disease is far more severe than that of ordinary erythematous lupus or even lupus exedens; there is very considerable pain and sometimes synovitis, there is high temperature, nervous symptoms which sometimes end in coma, and in not a few cases the result has been fatal. Cæsar Boeck saw two well-marked cases of this curious disease in Norway.

The acute form is, however, the exception. More often the disease persists with more or less frequent exacerbations, the face appearing as if affected with constant erysipelas. Here also the end is usually death from marasmus, or from an intercurrent disease.

I have myself only seen one example of this remarkable affection, which occurred in the practice of my friend Dr Cavafy at St George's Hospital. The patient was a woman between thirty and forty; the affection occupied not only the face, head, and neck, but the greater part of the back and trunk. It looked like erythema of a somewhat gyrate form and there was unquestionable scarring. The temperature was high and the patient succumbed to pneumonia.

Lupus marginatus is rather a variety of ordinary lupus than of L. erythematosus.

Rhinoscleroma.—This uncouth epithet was applied in 1870 by Hebra and Kaposi to a newly-recognised form of disease,—a hard, smooth infiltration or new growth of the septum of the nose and the adjacent tissues of the alæ nasi and of the upper lip. It has a general resemblance both to lupus and to syphilis, but differs from both in not being prone to ulceration, a characteristic which also at once distinguishes it from epithelial cancer. Mr Hutchinson has given a lecture on this affection (Lect. 27). He has not seen any case which corresponds with the fourteen or fifteen seen in Vienna, but thinks he has observed cases of lupus which by their unusual hardness and other characters approached rhinoscleroma. A few additional cases have been published in Germany, reference to which will be found at p. 496 of Hans von Hebra's 'Krankhafte Veränderungen der Haut.'

The ivory-like induration, the singular locality, and the absence of ulceration seem to separate it from ordinary lupus. Frisch has discovered a bacterium ('Ziemssen's Hbdh.,' xiv, 713), and since then Cornil and Alvarez have also seen it ('Ann. de derm. et de Syph.,' vi, No. 4, abstracted in the 'Lond. Med. Rec.,' August, 1885, p. 345).

The histological characters are not distinctive, for Kaposi found only infiltration of the cutis with very minute leucocytes. Geber recognised giant-cells and spindle-cells ('Arch. f. Derm. u. Syph.,' 1872). In a doubtful case brought by Mr Morrant Baker before the Pathological Society in 1881, Mr Hutchinson, Dr Cavafy, and myself were appointed a committee, and drew up a report which will be found at p. 262 of the report for that year.

A figure is given at page 458 of Ziemssen's 'Handbuch' by Schwimmer and Babes.

On the whole rhinoscleroma appears to be more nearly related to lupus than to any other disease.

It has returned after removal in cases reported from Germany and from Italy, but is said to have been favourably influenced by the application of salicylic acid in the belief that it would act as a germicide.

LEPROSY

(*LEPRA VERA—ELEPHANTIASIS GRÆCORUM*)

History and terminology—Geographical distribution—Anatomy and histology—The bacillus lepræ—Course and symptoms and event—Ætiology—Treatment.

This disease, so interesting from a historical point of view, is still of practical importance in many parts of the world; but I must here content myself with a very brief account of it, referring the reader for further information to the elaborate article by Kaposi, in Hebra's great work.

Nomenclature.—The name given to the disease by the Greeks was *lepra*, and it was divided into *alphos*, *melas*, and *leukos*. The disease, however, appears to have been rare in ancient Greece, and it seems to be not quite certain that the Septuagint translators were correct in translating *zaraath* of the Hebrew Scriptures by the Greek word λέπρα. The latter term, however, is universally applied to leprosy in the New Testament. It refers to the scaly surface often seen.

The Egyptian leprosy of Lucretius was quite as likely to be what we now know as elephantiasis (sc. Arabum):

> "Est elephas morbus qui propter flumina Nili
> Gignitur Egypto in media, neque præterea usquam."
>
> De rerum Nat., lib. vi, 1112.

Celsus, however, who describes *alphos*, *melas*, and *leuce* as species of Vitiligo (lib. v, cap. 19), clearly and distinctly portrays leprosy as a disease affecting the bones and the whole body, almost unknown in Italy, "*quem ἐλεφαντίασιν Græci vocant*" (lib. iii, cap. 25).*

In the Middle Ages leprosy was known to the school of Salerno as *mal morto* and *mal di San Lazaro*. The Arabic name of true leprosy is *Judzam* (*lepra Arabum*). *Barat* (= *leuce* = *vitiligo*) or "white leprosy" is nothing but leucodermia or deficient pigment.

The term applied by Willan and Bateman to leprosy was "elephantiasis Græcorum," while "lepra" was unfortunately used for part of the white scaly disease which the ancients would probably have recognised as alphos, and which all modern dermatologists call psoriasis.

No doubt many other cutaneous affections, obstinate chronic eczema, syphilis, lupus, and perhaps psoriasis, were confounded with leprosy in ancient times; but there is no question that one and the same destructive form of disease has existed in Palestine under the Mosaic Law, in Western Europe during the Middle Ages, and at the present day in many parts of the globe.

Distribution.—Norway is the only European country in which leprosy is still common; it is there known as *Spedalskhed.* It is also found in Sicily and Malta, in certain parts of Portugal, in the Levant, in the Crimea, and at

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* It must be remembered that the terms Elephas and Elephantiasis do not refer to rough skin or swollen and shapeless limbs, but to the magnitude of the disease. "Elephantiasis a magnitudine et diuternitate nomen accepit" (Aetius). "Est lepræ species elephantiasisque vocatur, Quæ cunctis morbis major sic esse videtur, Ut major cunctis elephas animantibus extat" (Macer). So also Aretæus.

Astrakan; also in Persia, Bengal, S. India, Burma, and Siam; in Japan and in China, where it is said to have been known for ages under the title *fa-fung*; in Egypt, Nubia, the Soudan, the Cape Colony (where it co-exists with elephantiasis Arabum), and most parts of the African coast (though apparently not common in the interior); in Madagascar and the Mauritius, St Helena, the Canary Islands, and the Azores; in Mexico and the West Indies (especially Trinidad), Central America, British Guiana, and Surinam, Bahia and the Coast of Brazil; in New Zealand, the Sandwich Islands, and some other parts of the Pacific.

Accounts of the disease from many of these and other places will be found in a valuable report on leprosy by the College of Physicians prepared for the Colonial Office and issued as a Blue-book in 1867. In 1874 Dr Vandyke Carter published an official report upon leprosy in India, and Dr Liveing treated the subject in his Gulstonian Lectures before the College of Physicians in 1873 ('Brit. Med. Jour.,' March 15th).

Varieties.—Leprosy is essentially one and the same disease, but two forms are usually predominant, the *nodular* or "tubercular," and the *anæsthetic*. The two are, however, usually combined. Either may be preceded or accompanied by pigment-spots, which have led to a third species being formed—*lepra maculosa*. All end in an ulcerative stage and all may lead to loss of members—described as *lepra mutilans*. "Black leprosy" is the only genuine form; "white leprosy" is not leprosy at all (p. 752).

Onset.—The disease begins insidiously, but in some cases with an outbreak of bullæ resembling those of pemphigus. There follows the appearance of red or violet patches varying from a finger-nail to the palm of the hand in size, which gradually become darker in colour. At the same places, or independently, appear flat, firm, raised nodules, consisting of an infiltration of the deeper parts of the skin. The lymph-glands at the same time enlarge. These nodules of tubercular lepsosy may shrink and be absorbed, leaving atrophied and sometimes pigmented spots; but more often they soften and ulcerate. The leprous ulcers, when once formed, secrete but little pus, and show few and feeble granulations. They slowly increase both in extent and depth.

Locality.—The leprous spots usually appear on the trunk, also upon the limbs and seldom on the face. The nodules, on the other hand, appear first and are most developed in the face, where they produce a singular deformity, which the ancients described as *leontiasis,* and which once seen, even in a drawing, is never forgotten. The disease also affects the neck, shoulders, back, chest, and abdomen, but is most frequent in the extremities, especially on the extensor surface. Tubercles occasionally occur even upon the palm and sole. The hands and feet are swollen and distorted, with thickened and rough skin, the ulcers burrow deeply and affect tendons, bones, and fibrous tissues, until at last toes, fingers, or the entire hand or foot undergo gradual necrosis and fall off.

The mucous membranes are also affected, particularly those of the mouth, nostrils, and larynx, also the conjunctivæ.

Moreover, the disease involves the great nerve-trunks, where the leprous nodules can often be felt during life.

Histology.—Careful microscopical investigations by Virchow, Thoma, and others showed that the disease consists in infiltration of the deepest layers of the cutis with granulation tissue. Leprosy was therefore classed by Virchow in proximity to lupus, from which, however, its clinical course, geographical distribution, and entire natural history widely separate it.

A bacillus was discovered by Hansen, of Bergen in 1874, which he described and figured in the 'Quart. Journ. of Micro. Sci.' for 1880 (vol. xx, p. 92). These microphyta appear to be of constant occurrence in leprous nodules; they have been found by Neisser, Cornil, Köbner, Dr Hillis ('Path. Trans.,' 1883, pl. xxii) and Dr Thin ('Med.-Chir. Trans.,' vol. lxvi, pl. xii, xiii) and by Dr L. J. Steven, of Glasgow ('Brit. Med. Journ.,' July 18th, 1885).

Course.—The progress of leprosy is extremely slow, and it resembles syphilis and lupus in producing very little pain. Patches of anæsthesia are sometimes found and may even be followed by ulceration before tubercles appear. It is said that as a rare occurrence hyperæsthesia precedes or takes the place of loss of sensibility. The anæsthetic spots usually show some amount of atrophy, the skin is paler than usual unless it be already the seat of pigment, and the hairs are small and deficient in colour.

While this terrible disease goes on its tedious though sure course, interrupted from time to time by temporary improvement and healing of the ulcers, but never more than checked, the general condition of the patient is wonderfully little affected. Even perspiration takes place very much as usual. The hair, however, is gradually lost, not only that of the scalp and beard, but the eyebrows and eyelashes. There is no fever, the temperature is usually subnormal, and the patient suffers much from cold. The pulse is slow, the appetite and other organic functions, including the quality of the urine, are very little altered. There appears to be no foundation whatever for the assertion of the ancient physicians that the sexual instinct is increased in lepers. It seems not unlikely that the name *satyriasis,* first applied to the distorted and hideous features of the sufferer, was afterwards misinterpreted, and thus led to the above assertion.

Death appears seldom to occur directly, for there is neither excessive pain nor hæmorrhage nor invasion of vital organs to hasten it, but when once fallen into a condition of anæmia and marasmus the miserable victims of leprosy are cut off by some intercurrent affection such as pleurisy, pneumonia, dysentery, or Bright's disease, all of which have been recorded in cases of leprosy by the Norwegian pathologists, Boeck and Danielsen, but none with sufficient frequency to show more than an accidental connexion with the disease.

Ætiology.—The essential cause of leprosy is entirely unknown. It has probably existed from the earliest times, and has only disappeared from civilized Europe within the last 400 years. We may hope that it is in slow but steady process of extinction in other regions. Notwithstanding the apparently constant presence of the *bacillus lepræ,* the disease is proved by the concurrent testimony of almost all competent observers to be non-contagious; it is not transmissible by living in the same house, by contact, or even by sexual intercourse. It is, however, possible that contact of actually ulcerating leprous nodules with a fissured skin or mucous membrane might produce the disease, and there is perhaps some reason to believe this possible contagious quality to be more marked when the disease is newly introduced, as it probably, has been in some of the South Sea Islands.

Whether or no under any circumstances contagious, leprosy is undoubtedly hereditary, so that its occurrence in persons of pure European parentage is excessively rare. In the few cases I have had under my care the patients have always been either half-castes or persons who were born and lived in India, and one of whose parents was probably of mixed blood.

An interesting theory has been put forth by Mr Hutchinson and defended with his accustomed ability. He thinks that leprosy depends in some way or other upon eating fish, probably fish in a state of decomposition. This view certainly agrees with its presence, not only on the sea-coast, but also in the neighbourhood of great rivers and inland lakes; and it also accords with the large consumption of salt fish in the Middle Ages, when it formed a principal animal food throughout the winter, as well as during Lent and other fasts. No undoubted proof, however, of the connection of leprosy with fish eating has been given. The disease does not appear in many parts where fish, both fresh and putrid, is largely eaten, and it is prevalent in certain districts where (so far as testimony can be trusted) fish do not form an article of diet.

Sex and age.—Leprosy appears to be somewhat more common in men than in women, in Bombay, according to Dr Carter, very much so. It begins usually about the time of puberty or in young adults. No congenital case, I believe, is on record. It is doubtful whether it has any predilection for castes or races as such, although at the present day it is, as above stated, almost confined to certain of the dark races of mankind, and where prevalent is rare among the well-fed and well-cared-for classes.

Treatment.—This is unfortunately almost hopeless, and we must rather look to the gradual rooting out of the disease by improved conditions of life than to any drug. Various remedies have of course been vaunted from time to time as specifics, but have all been in turn discredited. Cod-liver oil is the only internal remedy which can be said to do more than alleviate symptoms. Externally Gurjun and Chaulmoogra oils have been supposed to be valuable. I have tried the former in three cases with no benefit. Dr Liveing's much larger experience gives the latter a probability of being really of service.

Mr Hutchinson has recorded a case of gradual spontaneous recovery ('Med.-Chir. Trans.,' lxii, p. 331).

Leprosy is the only exotic disease of the skin which appears to demand separate notice.

Frambœsia or Yaws, apparently a contagious malady, and by some authors believed to be nothing but Syphilodermia, was known to Bateman and is described at length by Kaposi in Hebra's 'Handbook.' It is endemic on the west coast of Africa, but appears to be identical with what is known as *Pian* in Java and as *Verrugas* in Peru. Less clear is its relation to *Parangi,* endemic in Ceylon. *Radesyge* in Norway is, according to Hebra, lupus.

Aleppo evil.—This affection, known also as the *Delhi boil* and *bouton d'Aleppe,* has been ascribed to syphilis, but probably without reason.

Among the Erythemata ought probably to be included *Pellagra,* an epidemic disease observed in Lombardy, and probably connected with eating diseased maize, and *Acrodynia,* described by Alibert as epidemic in Paris during 1828 and 1829. Winternitz ("Eine klinische Studie ü. das Pellagra," 'Vierteljahrsschrift f. Derm. u. Syph.,' 1876) doubts the very existence of the former. Acrodynia appears to be endemic in the Levant, but its nature and causes are also obscure. (See Behrend, 'Hautkrankheiten,' pp. 154 and 156.)

Ringworm appears in peculiar forms in certain foreign countries. Burmese ringworm has been already referred to (p. 705); and Dr Anderson, of Glasgow, has published an interesting account, with figures, of *Tinea imbricata* from Tokelau, in the South Seas ('Edin. Med. Journ.,' Sept., 880).

TUMOURS OF THE SKIN

FIBROMA MOLLUSCUM.—*Distinction from molluscum sebaceum—Anatomy and distribution of the tumours—Their course and treatment.*

CHELOID.—*Terminology and history—Appearance, course, and symptoms—Histology—Diagnosis—Distribution—Prognosis and treatment.*

Xanthoma (see p. 280).—*Myoma—Neuroma—Angioma or vascular nævus—Elephantiasis teleangiectodes—Lymphangioma—Kaposi's xerodermia—Carcinoma, sarcoma, and rodent ulcer of the skin.*

Passing from the deep intractable ulcerations, combined with hypertrophic or neoplasmic processes, of lupus and leprosy, we come to the new growths or tumours of the skin in a more restricted sense.

The relation between the deep chronic inflammations of the skin, hypertrophy, and new growth is so close that lupus, tertiary syphilis, and leprosy might be classed either as deep destructive forms of dermatitis, or as cutaneous granulomata; while warts and condylomata, gutta rosea, and elephantiasis are as much new growths as inflammations. But we have now to treat of neoplasms in the stricter sense of the word, neither hypertrophies nor inflammations.

As in other parts of the body, the tumours of the skin are clinically "innocent," "malignant," or "semi-malignant;" while anatomically they are "homologous" or "heterologous" (*cf.* vol. i, pp. 81, 82).

FIBROMA MOLLUSCUM.—This affection of Bateman, the *molluscum pendulum*, also known as *molluscum fibroma*, differs altogether from molluscum contagiosum treated of above (p. 687), except in the fact that they both consist of multiple pedunculated tumours. Those of fibroma are not cystic growths, they are not glandular, and they have none of the histological characters of contagious molluscum. They are soft and painless, the skin over them is unaffected, they are more or less pedunculated, they vary in size from a pea to a marble or a fist, and when cut into they show œdematous, inelastic connective tissue. They are not unlike, both in appearance and structure, the firmer kinds of polypi of the nasal fossæ, the colon, rectum, uterus, and other parts of the mucous membrane. They might in fact be well termed "multiple fibrous polypi" of the skin.

The number of tumours is sometimes almost innumerable, as is seen in the well-known case of Virchow which forms the frontispiece to his work on morbid growths ('Kr. Geschw.,' Bd. i, S. 325). The size varies from a pin's head to a foot or more in diameter.

The celebrated case of Tilesius, of Leipzig, published in 1793, was named molluscum from the soft fleshy character of the tumours (*corpus tectum est verrucis mollibus sive molluscis*). There is no doubt, as Bateman saw, that these were not glandular and were quite distinct from the molluscum contagiosum described by himself. The skin of Rheinhardt, the Mühlberg peasant, who came under the notice of Tilesius, is still preserved in the museum of Leipzig.

Dr Fagge believed that these tumours begin in the outer sheath of the hair-follicles and sebaceous glands; and in one case he found an enlarged sacculated gland occupying the interior of one of the growths. This has, however, not been again observed, and the occurrence of similar tumours in the palm and sole seems to prove that the coincidence was accidental.

There appears to be little local predilection for these fibrous polypi. We sometimes see a single one on the face or elsewhere, or they may cover the face, the trunk, and the limbs. They also sometimes appear on the prolabium and the palate, as in a remarkable case described by Dr Fagge in the 'Medico-Chirurgical Transactions' for 1870, vol. liii, which was figured in Plate 18 of the Sydenham Society's 'Atlas' and modelled for the Guy's Hospital Museum (No. 497).

These fibromata occasion no pain, and single ones may be met with in perfectly healthy persons, to whom they cause no inconvenience. Some of these are congenital, but the typical multiple fibromata are certainly not so. They usually appear in childhood.

When they have attained their full growth they undergo no further change, and neither degenerate nor become absorbed. But, as Mr Hutchinson has pointed out, they sometimes lose their firm, fleshy feel and become flaccid so as to feel almost like empty cysts. (See the 16th of his Clinical Lectures "On Rare Diseases of the Skin").

Virchow has recorded a case in which the father, grandfather, and brother of a patient were all affected with multiple fibromata of the skin.

According to Hebra, when they are numerous the patient is usually ill developed in mind and body. But this is certainly not always the case.

Multiple fibromata are certainly rare, and probably few cases have failed of being recorded; but one or two polypi are not infrequently seen if looked for.

The only treatment is removal by scissors or the knife. The polypi show no tendency to return.

Cheloid.—In the 'Arbre des Dermatoses' of Alibert appears, among many other fantastic names, a new name for what was an undescribed disease —*Kéloide*. The etymology of the word was long a puzzle. It was supposed by some to be derived from κήλις, a mark, by others from κήλη, a tumour. Being taken by Addison in the former sense—*quasi ustione facta macula*— as meaning a scar from a burn, it was transferred to the curious affection still known as "Addison's keloid," but better named morphæa or circumscribed sclerodermia, described above (p. 723). It is now certain from the researches of Dr Fagge that Alibert meant by the word "kéloide" to denote the claw-like offshoots which characterise the disease in question, and intended to derive it from χηλή a crab's claw. The right spelling is now generally used, and Alibert's is recognised as the only "true" cheloid.

Alibert described it as "cancroide," and Bazin and other French dermatologists have hence called it malignant and regarded it as closely allied to epithelioma of the skin; but I think it not improbable that by "cancroide" Alibert did not mean "cancer-like" but "crab-like;" at all events it is not a cancerous or cancroid tumour in the modern sense of the word. Biett, Dieburg, Lebert and other writers have since published cases. Addison gave an excellent account of the disease in the 'Med.-Chir. Trans.' for 1854 (reprinted in his 'Collected Works' p. 170). Dieburg's paper was in the 'Deutsche Klinic' for 1852, No. 33.

The affection is a rare one; it consists of a fibrous or fibro-nucleated growth occupying the cutis vera.

It begins as a pink, smooth, slightly raised, flat nodule which increases in extent without becoming relatively more prominent. It is remarkably firm in feel. The centre becomes paler and is sometimes depressed, and the raised edges are surrounded by a slight erythematous border; the epidermis is completely adherent; it is in and not under the skin. Sometimes, however, especially in the later stages, it spreads to the subcutaneous tissue and forms adhesions to the deeper parts, but it never invades more than the integument.

The most characteristic part of the disease is the presence of radiated bands, which appear after a time, run across the original nodule, and afterwards project from its edge. These undergo contraction in the same way as the cicatrices of a wound, and the whole tumour is sometimes puckered and deformed by this process. In the earlier period the nodule might pass for a hypertrophied scar; in the later stages it still more closely resembles a large indurated and contracted cicatrix as from a deep burn or a syphilitic ulcer or a carbuncle.

The tumour is usually single but two or more may exist on the same patient. The disease is of very slow growth. It occurs most often in young adults of either sex. There is sometimes considerable pain associated with the growth of this curious disease and it is almost always tender. From the very commencement it is attended with pricking and itching with a sense of constriction or severe stabbing pains.

Histology.—Microscopic sections show that the epidermis is thin but otherwise unaffected, the papillæ are destroyed and the cutis vera and subcutaneous tissue occupied by bands of dense fibrous tissue which are quite indistinguishable from those of a true scar. As in all cicatrices the sweat-glands, hair-sacs and sebaceous follicles of the part are destroyed in the process. Dr Warren, of Boston, published a valuable histological account of cheloid in the Transactions of the 'k. k. Acad. d. Wissensch.' (Vienna, March, 1868). See also that by Babes in Ziemssen's 'Handbuch,' xiv, p. 434.

Diagnosis.—Neither in the histology nor in the symptoms does there seem to be any obvious distinction between a cheloid tumour and a hypertrophied and painful scar. Hebra, in fact, defines cheloid as an idiopathic or primary cicatrix. Others have maintained that all cheloid tumours are hypertrophied scars, and undoubtedly they can often be shown to arise from ordinary cicatrices or from the slight marks left after leech bites or acne pustules.

Dr Goodhart has published a remarkable case of cheloid growths following smallpox in the 'Clinical Transactions,' vol xiii, p. 51.

Dr Liveing, while admitting that cheloid growths often begin in scars, finds the distinction between them and hypertrophied cicatrices in two points; first, that the bands of fibrous tissue in cheloid run in definite parallel or radiating bundles, whereas those of a cicatrix form an irregular network; secondly, that the cheloid growth invades healthy tissues, which hypertrophied scars never do, and that this is the case even when cheloid appears in a previous scar. The new growth can be distinguished as it invades the old cicatricial tissue.

Locality.—Cheloid tumours occur most frequently in the skin over the sternum. They have also been observed on the abdomen, shoulders, arms, and face. They are usually single, and very rarely more than two, except in the case of cicatricial so-called false cheloid, when the new growth may appear in as many scars as were originally present.

Nature.—Pathologically we must, I think, consider cheloid not as a mere hypertrophy or a granuloma, but as a fibro-cellular new growth, a true sarcoma, sometimes consisting chiefly of spindle-cells, sometimes more exclusively of fibres. It has two characteristic marks of sarcoma apart from its histology, namely, that it is very apt to return again and again after removal, while on the other hand it does not reappear in the neighbouring lymph-glands or in the viscera.

A number of excellent models of cheloid, Nos. 454 to 466, were made by Mr Towne for the Guy's Hospital Museum.

I once saw a well-marked case of cheloid affecting the pubes in a patient of Mr Bryant, a man who probably had never had ulceration, syphilitic or other, of this part.

Acne cheloid has been described as a separate affection. It commonly occurs upon the shoulders where acne cicatrices are usually the deepest and most extensive.

Traumatic, or false cheloid, is better called a hypertrophied scar ("die warzige Narbengeschwülst" of Dieburg). It may occur whenever a burn, ulcer, or other injury produces a scar which then hypertrophies and becomes painful.

Prognosis and treatment.—Trustworthy observers have recorded the spontaneous disappearance of cheloid tumours, but this must be extremely rare. They seldom or never ulcerate. They grow slowly and appear not to menace life, but the pain they occasion is sometimes severe.

Unfortunately no treatment is of avail. If removed by the knife, by galvano-cautery, or by caustics, the tumours invariably return. Nor have any of the milder applications which have been tried produced absorption.

Mr Hutchinson, in an interesting paper on the subject ('Medical Times,' May 23, 1885), has recorded exceptional cases in which operation proved successful.

Xanthoma in the nodular form (*Vitiligoidea tuberosa* of Addison and Gull) is a true tumour of the skin, and therefore might find its place here. In its more frequent macular form (*Vitiligoidea plana*) it is rather a chronic deep dermatitis, resembling atheromatous inflammation of the arteries. The affection has, however, been already described as Xanthelasma in connection with jaundice (*v. supra*, p. 280).

Myoma.—Tumours of unstriped muscular fibre (Myomata, Liomyomata) have been described by Virchow ('Archiv,' vols. iii and vi), Klebs, Axel-Key, Rindfleisch, and Besnier ('Annales de Dermatologie' 1880). They probably take their origin in the muscular bands connected with the hair-sacs. They are of no clinical significance.

Neuromata, usually occurring as multiple, painful growths scattered over both trunk and limbs, have been long known and have lately formed the subject of a monograph by von Recklinghausen ('Über die multiplen Fibrome der Haut und ihre Beziehung zu den multiplen Neuromen,' 1882). Except in the pain which accompanies them, these tumours are indistinguishable from ordinary fibromata. Dr Duhring has described some severe cases marked by paroxysms of neuralgia.

Lipomata, or true fatty tumours, never affect the skin itself, but are always subcutaneous.

Vascular Tumour (*Nævus flammeus*).—More frequent are new growths of vascular tissue constituting nævi vasculares, angiomata or "mother's marks." Excluding moles or pigment-spots, these true or vascular nævi have always essentially the same structure. But they vary in appearance from the smooth, flat, "port-wine stains" as they are called, sometimes covering the greater part of the face, head, or even trunk, to the circumscribed pulsating tumour-like mass which can be removed by ligature, galvano-cautery or other mechanical means.

Of similar structure though different pathology are the stigmata of gutta rosea and erythematous lupus, and the permanently injected patches which sometimes accompany the cicatrisation of lupus exedens, syphilis, or any other deep form of dermatitis.

There are, however, some rare and remarkable forms of disease of the skin which, though anatomically angioma, differ from true nævi not only in being acquired instead of congenital, but also in their course and event. Sometimes they will, as described by Hebra, while spreading in some directions, return to a normal condition in others; or, again, they may acquire a tumour-like and semi-malignant character, growing rapidly and forming large masses of erectile tissue.

These are most often seen upon the extremities, though even here they are happily rare. They are sometimes complicated with fibrous growths, and these sometimes have not only the painful character of neuromata, but also the histological characters of that form of tumour. Brüns has described such cases on the lower extremities as "elephantiasis neuromatosa."

Under the somewhat similar title of *elephantiasis teleangiectodes*, Virchow and Kaposi have described multiple fibro-vascular growths, which begin as separate lobulated tumours, but afterwards form diffuse, vascular thickenings of the skin. Apparently identical with these is a case examined by Dr Liveing, and described and figured by the late Dr Tilbury Fox under the name of *fibroma fungoides* (pp. 352—354 of his work on 'Skin Diseases'). He there described other cases of fibro-vascular ulcerating growths which he considers of the same nature. One of these, however, may probably have been syphilitic.

LYMPHANGIOMA.—A curious affection of the skin which has been described under this name consists in a group of apparent vesicles, which, however, on careful examination, are found to be more deeply seated than usual, and in the event prove not to be inflammatory at all, but new formations, lasting unchanged for an indefinite period. In one case I have seen them strikingly resemble the vesicles of zona, appearing in several groups, and arranged in a tolerably regular line. In this case, the affection was complicated by appearing upon a large congenital port-wine stain, and the result was that many of the lymph-cysts became pink by admixture of their contents with blood, and when accidentally ruptured, thick red or black scabs were formed. This coincidence with ordinary vascular nævi (which others also have noticed) as well as histological investigation, prove, I think, that the disease is rightly regarded as analogous to acquired vascular nævi. But Mr Hutchinson has described the affection under, as I venture to think, the ill-chosen name of "lupus lymphaticus" ('Path. Trans.,' 1880, with fig.). Several cases have been brought before the Dermatological Society within the last few years. A careful histological description with figures by Mr Stewart will also be found in the 'Path. Trans.' for 1875, in the volume for 1879 (xxx, 474) by Drs T. and T. C. Fox, and in that for the following year (xxxi, p. 346) by Dr Sangster. Kaposi described a remarkable case of it in a woman twenty-two years old who had several hundred violet-red pimples, round or oval in shape, some of them as small as a lentil, situated in the cutis and somewhat resembling certain forms of syphilodermia. A minute portion being excised, showed that the cutis was filled with dilated lymph-spaces lined with endothelium. He named it *lymphangioma tuberosum multiplex.*

XERODERMIA (of Kaposi).—Perhaps the most remarkable of all cutaneous diseases, one which is at once allied to nævi in its early stages and markedly malignant in its later development, is a rare affection, first described by Kaposi in 1870 under the most unfortunate title of xeroderma, a name which had been previously applied to a totally different condition by the late Sir Erasmus Wilson, whose works are well known in Vienna. It has also been called "xeroderma pigmentosum," "Kaposi's disease," "angioma pigmentosum et atrophicum," and "atrophoderma pigmentosum."

It begins with spots of erythematous appearance not unlike those of measles. These fade and form pigment-spots like freckles. The third stage may be months or years in appearing. The apparent ephelides then become atrophic, the skin dry, thin, and wrinkled. Next it gradually contracts, so as to form a smooth, tightly-drawn surface, which may evert the eyelids or the lips or contract one of the joints. At the same time fresh brown pigment-spots and stigmata appear on the affected surface. The former undergo the same atrophic changes, the latter may increase until they resemble congenital vascular nævi.

The disease is not accompanied with itching or pain, yet after continuing in this comparatively innocent form for months or sometimes years, the vascular spots begin to become warty and to ulcerate. Fungoid growths of a most malignant character appear at last, not only in the maculæ, but also in distant places, and death ensues by hæmorrhage or exhaustion.

Hebra and Kaposi together observed only four cases of this remarkable affection. Erasmus Wilson described another under the name "general atrophy of the skin." A remarkable case was shown at the Clinical Society as one of lupus, and was recognised as identical with Kaposi's disease by Dr T. C. Fox. This same case, with the others in the same family, will be found fully described by Dr Crocker in the 67th volume of the 'Medico-Chirurgical Transactions,' p. 196, with coloured lithographs, and a table of thirty-four recorded cases. These were recorded by Rüder in a monograph on the subject, by R. W. Taylor, of New York, by Neisser, and by Vidal.

From this table it appears the disease has never been yet observed above the age of puberty. One case occurred in an infant four months old, most under two years, one at nine, and one as late as sixteen.

It occurs in boys and girls indifferently and most frequently more than one case is found in a family. Twenty-six of the thirty-four cases in Dr Crocker's table belonged to nine families.

The histological characters of the spots are those of vascular dilatation, of pigmentation, and of atrophy. The final tumours appear to be always true epithelial carcinoma, not sarcoma.

Treatment has at present been unavailing.

The ordinary malignant growths of the skin are happily infrequent, nor have they many special points of interest; for their pathology is essentially the same as that of the corresponding growths upon mucous membranes; moreover, their recognition is not difficult and their treatment purely surgical, so that but little need be said of them in this place.

Carcinoma fibrosum, or scirrhous cancer, the most typical of all the forms of cancer, rarely affects the skin primarily, though it frequently infiltrates it as the result of primary carcinoma of deeper parts, as, for instance, of the mamma. I have seen three examples of the remarkable form of hard, indurating, and widely-spread cancer described by Velpeau as *squirrhe en cuirasse*. One was a patient of Velpeau's in whom the disease had spread from a cancerous breast; another was a patient of Dr Humphry's, of Cambridge. The remarkable and wide spread induration before ulceration begins and before implication of deeper organs occurs, renders it peculiar and produce a superficial resemblance to sclerodermia or to rhinoscleroma or certain forms of lupus. It is usually secondary to mammary cancer.

Epithelioma, or keratoid cancer (see vol. i, p. 101), is the most common form of malignant disease in the skin. Even this is rare, compared with its frequency in the œsophagus and large intestine, and at the labial, anal, and urogenital orifices. Its formerly most frequent seat, the scrotum, is happily no longer so, and "chimney-sweep's cancer" has become a rare curiosity in this country.

The term "epithelioma," first applied by Hannover, of Copenhagen, to this disease, of which he was the first to describe the histology, was discarded by Virchow for "epithelial cancer." Unfortunately "epithelioma" is now applied by some German writers to Molluscum contagiosum.

Rodent ulcer.—This affection, originally described by Jacob, of Dublin, is now ascertained to be histologically carcinoma. (See Mr Hulke's paper in the 'Path. Trans.,' vol. xxii.) The presence of epithelial cells in the cutis vera and of the nest-cells characteristic of the horny form of cancer leave no doubt of its real pathology. It is, however, the least malignant of cancerous growths, for it spreads slowly, there is little new growth, and it rarely affects even the neighbouring lymph-glands. It is usually seen near the eye, upon the side of the nose, on the cheek, or the temple. Like other kinds of carcinoma, it is a disease of mature life or of old age. Its early stages are those of a small, smooth, pale growth not unlike a wart. If, as is sometimes the case, it has begun in a congenital mole, it retains the pigment of that structure. It often has a pearly aspect, so as to look somewhat like a molluscum tumour or even like the cysts not unfrequently found about the eyelids. When ulceration begins it is covered by a rather thin, dark and adherent crust. It produces little or no pain and advances so slowly that when it first comes under the surgeon's eye it presents the appearance of a chronic, indolent, indurated ulcer with sharp, well-defined, nodular edges, and no granulations. In its later stages it resembles more nearly its pathological allies, epithelial cancer of the lip, the scrotum, the glands, or the vulva.

Other cases clinically of rodent ulcer appear to have a different histological structure (Verneuil, 'Arch. gén. de Méd.,' 1854, ii, 458, and Thin, 'Path. Trans.,' 1878, pp. 237, 241). A review of these and other papers from Thiersch downwards, by Dr Hume, of Newcastle, with histological drawings, will be found in the 'Brit. Med. Journ.,' Jan. 5th, 1884.

The diagnosis from tertiary syphilis lies in the ulcer being single, in its not invading the bones or other tissues, and in there being no other sign of syphilitic disease. From lupus it is distinguished by the scab being thin and dark, by its beginning at a much later period of life, and in the last resort, by microscopical examination of the material obtained by scraping or squeezing the edges of the ulcer. See also p. 729.

Sarcoma.—Beside carcinoma in the proper sense of the word, the skin is occasionally liable to multiple sarcomata. These are almost always secondary to some internal growth; by their large number, small size, and hæmorrhagic or sometimes melanotic character they may resemble certain forms of purpura or pigmentation. Cases of this remarkable affection have been already described in the first volume, pp. 95, 96.

PIGMENTAL, HÆMORRHAGIC, AND NEUROTIC AFFECTIONS OF THE SKIN

Albinism—Leucodermia, congenital and acquired—Its relation to leprosy—The state of the skin—Canities—Melanodermia, secondary to inflammation, syphilis, adrenal disease, malaria, &c.—Ephelis—Lentigo—Chloasma—Relation of melanodermia to leucodermia—Petechiæ and vibices—Peliosis rheumatica—Pruritus—Trophic neuroses—Zona—Area—Leucodermia—Symmetry in cutaneous diseases.

Anomalies of Pigmentation.—We are familiar with degrees of pigmentation of the skin not only in different races, but also in the wide difference between individuals belonging to the same stock and even to the same family.

Albinism, or complete absence of pigment, not only from the skin and its appendages, but from the iris and choroid, is always a congenital defect in the human race, as in rabbits, mice, horses, and other animals. The so-called "white" elephants are either albinos or piebald.

Albinos occur occasionally among the dark races. The "white" negroes have a dirty pale skin, colourless hair, and pink irides with dark red pupils.

Partial albinismus—Leucodermia—Vitiligo.—This also may be a congenital variety or "malformation." Piebald horses may be called "abnormal," but we should scarcely say so of cattle, dogs, swine, or guinea-pigs. This condition is, however, far more common in domesticated races than in a state of nature.

A similar congenital "piebald" state of the skin is occasionally seen in human beings. In negroes and in the Indian population it appears to be not uncommon. We sometimes see it in this country, as white locks of hair.

When acquired after birth, leucodermia has been, and still is, confounded with leprosy. In fact, "white leprosy," when it does not apply to psoriasis, seems generally to mean leucodermia occurring in patches.

Celsus, lib. iii, c. 25, distinguishes elephantiasis (*i. e.* leprosy) from *vitiligo* (calf's skin, parchment skin), which he divides into three species (lib. v, c. 19):—*V. alphos*, scattered, colourless, slightly rough patches; *V. melas*, pigment spots, to be presently mentioned under melanodermia; and *V. leuce*, still whiter than alphos, with white hairs growing on the patches. But later writers speak of vitiligo,* and more particularly of leuce, as varieties of leprosy. The same explanation appears to apply to the Arabic term "Baras," the equivalent of Leuce or Alphos in Greek and Vitiligo in Latin, which was also intended to denote a white leprosy. The confusion is due to patches of skin occurring in true leprosy, which are either deeper or paler in tint than the surrounding surface. The ambiguity appears still to exist not only among the natives of Southern India and Ceylon, but among some physicians, judging by their reports in the Blue-book referred to before as published in 1867. Dr Vandyke Carter states expressly that leucodermia is commonly confounded with anæsthetic leprosy.

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* The term vitiligo has been also applied to a circumscribed smooth white indurated spot level with or slightly sunk below the surface. This would make it identical with morphœa, *i.e.* with circumscribed sclerodermia (*v. supra*, p. 723). The term may well be abandoned.

The skin in leucodermia is perfectly normal except for the loss of pigment. The Malpighian layer and also the hair are affected. There is no anæsthesia. The border is convex, and often a pigmented line separates it from the normal skin around. This was the case in the specimen which Gustav Simon first examined histologically. The patches are usually multiple, sometimes very numerous. They are occasionally symmetrical,* more often irregular, with no predilection for one surface of a limb or the other. They may occur anywhere, but are most frequent on the trunk, especially the abdomen and genitals, where natural pigmentation is deepest.

Leucodermia is more common in hot countries and in the south of Europe than in England, but here cases are readily overlooked, since in most cases they are inconspicuous, and give rise to no discomfort.

Removal of the white patches has been attempted by blisters and other irritants, and also by tattooing. The result is not often satisfactory.

Canities.—General blanching of the hair is a well-known senile change. But, like baldness, it often occurs in early adult life, especially when the hair is very dark. Besides suffering loss of pigment, which gives the dull, yellowish, "milk-white" appearance, the hair is apt to become dry and admit air-bubbles, which increase its refractive power, and produce the glistening steel-grey or "silvery" aspect.

Many instances are on record of rapid blanching of the hair of head or face in consequence of mental anxiety or grief. The cases of Sir Thomas More, of Henry the Fourth of France, of Marie Antoinette, have become historical, and the fact of this premature senile change coming on in the course of a few hours seems impossible to deny. Bichat and Alibert record cases which they actually saw, and Brown-Séquard has seen rapid blanching in his own beard ('Arch. de Phys.,' 1869, p. 442). A grey patch sometimes follows neuralgia (*cf.* vol. i, p. 671). The late Dr Laycock quoted an instance in which a sepoy was seen to turn grey in half an hour ('Med. Times and Gaz.,' 1862). I once met with a case myself. A young man consulting me for some slight ailment had perfectly white hair. In answer to my inquiry he stated that a few years before he had fallen asleep after a debauch, and on waking in a cold room in the morning found his hair had turned white. I objected that his beard was brown, but was told that when the change of colour occurred it had not yet grown. This sudden change is probably due to development of air-bubbles.

Melanodermia—Melasma cutis, cloasma.—Increased pigmentation of the skin, like its diminution, may occur either universally or in patches.

The former condition is never congenital like albinismus. A dark skin at birth is always hereditary. It may occur as the result of exposure to the heat of the sun or to other irritants, or as the result of certain internal diseases.

As the result of hyperæmia, or slight superficial inflammation ("eczema solare"), one sees increased pigmentation produced by the wind in cold weather, or in driving, or by the cold of snowfields, which, as Alpine climbers know, will scorch the face without sunshine. I remember seeing among Professor Hebra's patients a youth who had wandered over a great part of Hungary in the depth of winter and in rags. The exposed parts of the skin had become almost the colour of a mulatto, yet there had been little or no sunshine.

Although all hyperæmia produces more or less increased pigment, there is considerable difference in different inflammatory diseases. The deeper and more chronic forms of dermatitis have very little effect, as we should anticipate from their primary seat in the cutis vera. Chronic eczema and chronic traumatic inflammation produce great pigmentation, as seen in the brown, almost black patches which surround indurated varicose ulcers in old people. Ordinary eczema may last, however, for a very long time without producing much darkening, and the same is true of impetigo and scabies.

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* See a remarkable case of perfectly symmetrical leuco- and melanodermia figured by Dr Lesser (Ziemssen's 'Handbuch,' Bd. xiv, 2te Hälft., p. 186, fig. 11).

Of the superficial inflammations, chronic inveterate prurigo produces perhaps the greatest pigmentation, and the same is the result of prurigo pedicularis, aided probably by the scratching which it occasions and also by the age of the patient; for all pigmentation is slow in childhood and rapid in old age.

Certain forms of erythema are accompanied with increase of pigment, particularly pellagra (acrodynia), and urticaria pigmentosa (p. 754).*

Psoriasis very early and readily causes pigmentation, and the colour is sometimes quite indistinguishable from the coppery hue of a syphilitic eruption. Indeed, we may say that next to syphilis, psoriasis will produce pigmentation in the shortest time.

Besides the well-known brownish pigment which gives its characteristic colour to even early forms of specific eruption, a somewhat rare form of syphilis has been described by French authors as the "café au lait" form of syphilide. I have seen cases of it, both in Paris and in London, in the form of ill-defined brownish maculæ occurring on the neck of women who were the subjects of secondary syphilis (*cf.* vol. i, p. 120).

Pigment has already been mentioned as occurring in some cases of sclerodermia and in the malignant kind of atrophic nævi of the skin called "xerodermia" by Kaposi (p. 749). The remarkable increase of pigment in the course of Addison's disease has been fully described in the chapter on that subject. Increased pigmentation, though far less intense, is observed as the result of malaria, and to a still slighter degree in the cachexia of internal cancer.

Maculæ—Ephelides—Lentigo.—It remains to mention circumscribed pigment-patches, which occur without inflammation and independently of any other morbid sign. The most familiar are the small, dark brown or yellowish spots which, when they occur on the face, are named freckles (*ephelides*). They are no doubt the result of exposure to the sun. They occur most frequently on the face, but also upon the hands and arms when these are bare. They are almost confined to xanthachroic complexions, and are particularly common in persons with red hair, blue eyes, and the delicate pink and white skin which so often goes with them. These freckles also, like the diffuse pigmentation of sunburn, disappear in time, though much more slowly.

Precisely similar minute dark spots occur in covered parts of the skin, and in mucous membranes, sometimes along with the melasma of Addison's disease or with pigmentation from malaria, and sometimes in conditions of health. Others are congenital and may then be described as pigmentary nævi or "mothers' marks." When combined with a congenital papillary growth, often covered with a strong growth of hair, they are called "moles."

Chloasma.—More diffused and less intense patches of pigment occur upon the forehead of pregnant women, and have long been known under the name *chloasma uterinum.* In some cases they appear during each pregnancy and disappear after delivery. The word chloasma was at one time extended to the pigmented patches on the trunk which we now know to be due to a fungus and call tinea (or pityriasis) versicolor. But there seems no reason why at present the term should not be reapplied in its original signification.

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* I take this opportunity of referring the reader to the best account yet published of this curious affection, with a histological examination of the skin, and a table of nineteen recorded cases. It is by Dr Colcott Fox, and appears in the 66th vol. of the 'Med.-Chir. Trans.,' p. 3

Similar pigment-spots on the forehead and about the eyes are symptomatic of ovarian irritation, and appear in some cases of dysmenorrhœa with each menstrual period. (See eight cases reported by Dr Champneys with valuable comments in the 'St Barth. Hosp. Rep.,' vol. xv.)

Such pigmentation may also be the result of sexual excesses in male subjects, but this cannot be distinguished from the dark circles round the eyes which often accompany severe attacks of headache, especially megrim. All these cases may be grouped together by their clearly neurotic origin. They must be carefully diagnosed from not unfrequent instances in which lampblack or other pigment has been designedly applied to the face, forehead, and eyes by hysterical or otherwise deceitful women.

Lastly, there are certain cases in which patches of pigmentation occur in various parts of the body, unconnected with local irritation and without any internal disease.

These cases of idiopathic circumscribed melanodermia are decidedly rare and are generally associated with leucodermia. White patches occur in the pigmented surface. The former are sharply defined and have convex borders, the dark surface is most marked close to the white (allowing for the effect of contrast) and gradually shades away into the normal skin. Most cases, as far as I have observed, may be called either melano- or leucodermia, or both at once; and apparently consist in an irregular distribution of pigment. The white patches come usually first.

Solutions of corrosive sublimate, such as "virgins' milk" and "Gowland's cosmetic" (p. 683) are believed to have the power of removing freckles. The mingled patches of white and dark skin just described are best left alone, but circumscribed pigmentary nævi which cause disfigurement on the face may, if small, be removed by excision or galvano-causis.

Cutaneous hæmorrhage.—The most important conditions in which ecchymoses, whether the small ones like fleabites (hence called *petechiæ*) or the larger ones named *vibices*, are seen upon the skin are those of scurvy and of purpura. In both cases hæmorrhage occurs in other parts as well as the cutaneous surface, and in scurvy the ætiology of the disease serves to define it. Both affections have been already treated at length in this volume (pp. 577, 605).

It only remains to mention the principal cases—besides ecchymosis from direct mechanical injury—in which hæmorrhage accompanies cutaneous diseases.

The peculiarities of hæmorrhagic smallpox have been already described (vol. i, p. 224 and also p. 231).

In eczema, scabies, psoriasis, lichen and prurigo, ecchymoses never occur except as the result of scratching. Slight hæmorrhage often tinges the contents of the bullæ of pemphigus, especially in the gangrenous, cachectic, or "malignant" form of the disease; but I believe it does not occur either in Pemphigus (*vel* herpes) gestationis or in Pemphigus foliaceus.

All forms of erythema are liable to be complicated with hæmorrhage. It is rarest in urticaria (when it constitutes the Purpura urticans of Willan), very common in erythema nodosum, when it produces the subsequent bruiselike pigmentation, and most frequent in the forms of erythema which occur in the course of rheumatic fever. This last condition seems to have been first observed by Schönlein, who named it *Peliosis rheumatica.* The erythematous patches appear acutely, with fever and synovitis. They are most often seen on the back of the hands and feet, the forearms and shins, but may also affect the thighs, hips, and trunk; they are not often symmetrical. Either from the beginning, or soon after the appearance, the redness is found no longer to fade on pressure; hæmorrhage has taken place. Successive crops of these papules or large patches may occur, each lasting about a week, and disappearing with only a slight macule to mark its place.

I do not think we need a special name for this disorder. It is a true erythema, whether occurring in the course of rheumatic fever or in persons who have already suffered from that disease, and we have seen that both this connection and liability to hæmorrhage are characteristic of the whole group of erythemata. On this subject see "A Case of Rheumatic Purpura," with notes by Dr Wickham Legg ('St Barth. Hosp. Rep.,' vol. xix.)

Neurotic affections of the skin.—The only cutaneous lesion which is certainly related to nervous disturbance is zona or herpes zoster, which has been described among diseases of the peripheral nervous system (vol. i, p. 366).

The sensory nerves of the skin lead by the medium of pruritus to scratching, and how important an agent this is has been shown in our accounts of eczema, prurigo, scabies, urticaria, &c. These pruriginous diseases are in striking contrast to syphilodermia and lupus.

But it has been supposed that disorders, not of sensory, but of trophic nerves produce cutaneous diseases other than zona. The existence of trophic nerves is a physiological fact, but their presence gives so easy an explanation that we must beware of admitting it without adequate proof.

Area has been referred to a neurotic cause, but I fail to see any proof of it, though supported by Dr Liveing and other good pathologists. There is little if any anæsthesia to be detected and the patches do not follow the course of cutaneous nerves, nor is there any other evidence of the nervous system being affected.

Leucodermia has been referred to the same cause, and Mr Hutchinson has advocated this view. But here again there does not seem to be any reason but the difficulty of finding a better "explanation" for ascribing the disease to nerves. All that can be said in favour of the neurotic origin of morphœa will be found in the oft-quoted 'Clinical Lectures,' vol. i, p. 313.

The formation of the wheals of urticaria may be ascribed to vaso-motor nerves, and it sometimes follows mental emotion with great rapidity.

Certain bullous eruptions appear to be connected with neuroses, and some writers, as Auspitz and Leloir, have endeavoured to distinguish true inflammatory vesicles or bullæ, with loculi, seated in the Malpighian layer, from subepidermic bullæ.

Some authorities believe that symmetry points to a nervous origin of a cutaneous disease; but others hold symmetry to be the mark of "blood diseases" and asymmetry that of neuroses. Neither belief seems to be well supported. Symmetry points neither to a constitutional nor to a blood disease. All general diseases are symmetrical because the human body is so; a one-armed man would be unsymmetrically affected by scarlatina or psoriasis. Again, psoriasis and eczema are symmetrical because they affect the skin of a certain structure and surroundings which is found on the corresponding parts of the limbs, ears, and other parts. But "diseases of the blood" like leuchæmia and purpura are not usually symmetrical; and "diseases of blood and bones," as Sir William Gull calls them, which affect not the blood only but all the living tissues, such as syphilis and leprosy, are scarcely symmetrical at all. The only nervous disease of which we are sure—zona—is, like neuralgia, markedly unsymmetrical.

See, however, on this subject a valuable paper by Dr Crocker, with numerous references ('Brain,' October, 1884, p. 343), and the monograph by Leloir, 'Recherches sur les Affections cutanées d'origine nerveuse,' 1882.

PRACTICAL REMARKS ON THE CLASSIFICATION AND DIAGNOSIS OF CUTANEOUS DISEASES

In the preceding chapters I have only occasionally discussed in a formal manner the diagnosis between two diseases which may be mistaken one for the other. This was partly because, if the characteristic symptoms and course of a malady are duly described, they form the real and only element of its diagnosis; and although it is a useful exercise for a student to make lists of the distinctive characters of two or more diseases, the attempt to fix them in a tabular form is of little service to others, and perhaps tends to foster artificial memory of words rather than to help familiarity with things. Symptoms differ endlessly at the bedside and none of them can be really considered what is called pathognomonic. Another and special reason is that the diagnosis of cutaneous diseases often turns upon very slight differences of the lesion or of the distribution which it is impossible to put into words. Moreover, much of what is called diagnosis is not distinction between one pathological condition and another, but only between certain more or less arbitrary forms which have been fitted with still more arbitrary names.

I propose in the present chapter to speak briefly of this question of diagnosis on which in its true meaning all successful treatment must rest, and particularly to point out some distinctive characters of syphilitic disease of the skin, which has not called for special treatment in this part of the work, since it has already been fully dealt with in the first volume (p. 116, *et seq.*)

Although it is desirable to distinguish and accurately to name most of the diseases which I have separately described, yet even in this list, which is far shorter than that met with in many treatises, there are not a few diseases which are so rare that they rank as little more than as curiosities. Such are Pemphigus foliaceus, Urticaria pigmentosa, Favus, and Xerodermia maligna.

Many important diseases of the skin again are exotic and only of practical importance for English readers who may practise their profession in India or the colonies. Of these my own knowledge is far too small to offer anything worth repeating, and even such affections as Leprosy and Lichen agrius must be learnt from personal experience and perusal of the descriptions of those who have seen and studied them.

Again, there are certain affections which, differing by more or less important characters in appearance and in histology, nevertheless agree very closely in their general pathology, in their causes so far as they are known, and, what is most important, in the kind of treatment which is generally suitable.

From a practical point of view, looking chiefly to questions of prognosis and treatment, it will be found that we may divide diseases of the skin into the following important groups for diagnostic purposes:—

I. *Factitious eruptions.*—We must never forget the possibility of the affection before us being factitious. All kinds of inflammation, eczema, erysipelas, pemphigus, impetigo may be simulated by the application of various irritants. Pigmentation also has been often simulated with success. Such artificial dermatites will generally be found upon the hands and arms, rarely on the face, and scarcely ever beyond reach of the patient's hands. The persons who are guilty of such attempts at imposition are usually either prisoners in gaol or those who from whatever cause are malingerers, or else they are hysterical, neurotic young women, girls, or boys. When one's suspicions are once awakened it is rarely difficult to detect the imposture. Mustard, cantharides, and some other irritants can be distinguished by the help of the microscope.

II. *Traumatic eruptions.*—In all cases of dermatitis we should seek for the irritant, and sometimes it is so directly the cause of the disease that the eczema or impetigo in question may be considered purely traumatic, and efficient treatment immediately follows accurate diagnosis, *sublata causa tollitur effectus.*

Pediculi in the hair should be carefully looked for in all cases of impetigo in children, pediculi vestimentorum in old people. Scabies itself is but an extremely definite and well-characterised dermatitis resulting from the presence of a living source of irritation. But beside these well-known cases, already fully described, of parasitic dermatitis, it will be found that some supposed cases of purpura in children are nothing but petechia produced by fleabites, resemblance to which indeed gave it the name petechia. Moreover, many cases of infantile prurigo, urticaria, and ecthyma are due to the presence of bugs or gnats. In adults pediculi pubis may sometimes be found in the axillæ as well as in their proper region, and when destroyed by mercurial ointment the patient is at once relieved from pruritus.

In many trades an irritant must be sought in the objects which the patient habitually handles. The coarser kinds of brown sugar are a frequent cause of eczema of the hands (grocers' itch). So with many of the "chemicals" used in a variety of modern handicrafts. Constant wetness of the hands in washerwomen, in scrubbers, in potmen and many other occupations produces eczema rimosum. The heat of the sun is the cause of eczema solare and ephelides, the heat of the fire of the pigment-spots on the shins of elderly people.

Sweat again is a very common irritant, producing the erythema which usually accompanies sudamina and also intertrigo of opposed surfaces. Scratching as a cause of traumatic dermatitis has been repeatedly referred to.

III. *Febrile rashes.*—We must take care never to forget the possibility of a cutaneous eruption being part of an acute exanthem. The use of a clinical thermometer is a great help in this respect, but I have seen a man with typhus (and the rash fully out) appear as an out-patient for a skin disease, and modified variola and varicella are both not unfrequently mistaken for acne or impetigo.

IV. *Syphilodermia.*—When we have satisfied ourselves that the eruption before us is not factitious, nor directly traumatic, nor a symptomatic eruption, we may next consider whether or no it is due to syphilis. In this inquiry it is undesirable to ask questions, the answers to which are as apt to mislead as to guide aright.

(1) We should first consider the colour of the affected skin, remembering however, that the pigmentation which gives the so-called coppery or raw

ham tint to a syphilitic eruption is the same which is sooner or later produced by all forms of dermatitis. Psoriasis, chronic eczema, lichen planus, and prurigo may all produce shades which bear the closest resemblance to syphilodermia.

(2) The lesions of syphilis are multiform or polymorphic. It is rare in any but syphilitic affections to find mere hyperæmia in one part and associated pustules, papules, scales, or ulcers in others, and it is not often that a syphilitic eruption exhibits only a single elementary lesion.

A pustular eruption in an adult should always suggest the question of syphilis when that of scabies has been answered in the negative.

(3) Syphilitic eruptions for some unknown reason do not itch, and the exceptions to this rule are remarkably few. These usually occur during the stage of scabbing of pustular rashes or during the healing of tertiary ulcers. I have, however, seen an ordinary secondary syphilide so irritable that wheals and scratch-marks were produced. On the other hand, psoriasis is often free from irritation, while the degree of itching of eczema and even of scabies and prurigo varies greatly.

(4) The local distribution of syphilitic diseases is a great aid in diagnosis. Specific eruptions are certainly not, as is often stated, symmetrical; the early roseolous rash is so only because it is general, and therefore, upon a symmetrical surface like the human body, more or less symmetrical. Moreover, as it chiefly affects the face, chest, and trunk generally, it is near the middle line; but we do not see symmetrical patches of syphilide in corresponding parts of both sides of the face, both sides of the trunk, or the right and left limbs. Moreover, in all but the earliest syphilides the affected patches are very decidedly and constantly unsymmetrical, irregularly scattered over head, trunk, and limbs, and chiefly remarkable for having no well-marked seats of predilection. The forehead, especially about the roots of the hair, is, however, very frequently the seat both of the early and middle erythematous, scaly and pustular syphilides, and the palms of the hands and soles of the feet are frequently symmetrically affected with the later scaly eruption.

Practically, when we find a disease of the skin occupying some unusual position, we should at least consider the question of syphilitic origin.

(5) These signs alone or in combination serve to distinguish the early roseola from erythema, eczema, scarlatina, and measles, and the somewhat later secondary eruptions from eczema, lichen, scabies, impetigo, and psoriasis.

The eruptions of *congenital syphilis* which are most liable to be mistaken are—the so-called pemphigus of infants, which is known by its affecting the palms and soles; rupia, which by the form of the crusts and the ulcerated surface beneath may always be distinguished from impetigo; an erythematous rash of the nates and genitals of infants, which is distinguished from eczema of the same parts, also common at that age, by its coppery colour, its blotchy distribution, and more defined margin.

The tertiary ulcers of syphilis are distinguished by their appearing on unusual places, by their punched-out edges, circular or so-called horseshoe shape and by their usually producing very little pain or discomfort, although as with itching this character is occasionally present. Tertiary ulcers have no predilection for the outside of the leg, but inasmuch as the part above the inner ankle is for anatomical causes the chosen seat of varicose ulcers, most ulcers in the first position will be syphilitic, and in the latter not. For the same reason most ulcers on the arms are found to be tertiary.

V. *Tineæ.*—The next great group of skin diseases will be those which are due to vegetable parasites—tinea versicolor of the trunk, eczema marginatum of the perinæum and thighs, tinea circinata of the neck and other parts, tinea sycosis of the chin, and tinea tonsurans of the scalp. Here the general characters detailed in the chapter on the tineæ are generally sufficient to show the nature of the affection to a practised eye, but in all doubtful cases the microscope should be employed.

Tinea of the scalp is rare in adults, and tinea circinata still more so; tinea marginata occurs only in adults, and, so far as I know, in males.

VI. *Superficial affections.*—To distinguish the superficial from the deeper kinds of dermatitis we should notice whether the cutis alone is infiltrated and thickened or whether it is bound down by adhesions to the subcutaneous tissues. The presence of scars, however slight, is a proof that the process has gone deeper than the papillæ and has more or less extensively destroyed the papillary layer. Superficial inflammations, excluding those due to the acarus, to pediculi, and to other direct irritants, and excluding those which are the result of vegetable parasites and of syphilis, fall with respect to their treatment into three large groups:

(1) The first, represented by impetigo and most forms of eczema, are subacute and accompanied with burning, itching, and pain, sometimes with a slight degree of fever. They are to be treated by local remedies designed to reduce the hyperæmia, diminish the exudation and calm the irritation, aided by light diet, free diluents, laxatives, and diuretics. In short they are to be treated according to the modern form of the antiphlogistic method.

(2) The second group of superficial inflammations of the skin is typically represented by psoriasis, but includes lichen planus, many of the more chronic, dry, and obstinate forms of eczema and true prurigo. They are best treated locally by tar or allied preparations, internally by arsenic. They are chronic with little irritation, exudation, pain, or signs of active inflammation.

(3) The third group is that of erythemata as defined above (p. 667). Here the indication is to correct some internal disorder of which the eruption is the symptom.

VII. *The acne group.*—Acne, both in its pathology and ætiology, differs from other forms of dermatitis. The age of the patient and its distribution are sufficient for diagnosis. It is at once a superficial and a deep dermatitis and is often followed by scars, but its treatment consists entirely, or almost entirely, in local applications directed to the correction of the sebaceous affection. With acne may be classed Sycosis and Furunculus.

VIII. *Deep affections.*—When we have ascertained that the affection of the skin is deep, that is to say that it goes below the papillary layer, the field for diagnosis is limited.

Excluding erysipelas, which is distinguished by its acute character and febrile symptoms, and excluding the pustular affections which affect the skin deeply and produce scars only at isolated points, such as acne, variola, and zona, and excluding, lastly, leprosy and other exotic diseases—we have to distinguish, in the great majority of cases which come before us in this country, first, traumatic and varicose ulcers; secondly, gummata and tertiary syphilitic ulcers; thirdly, lupus; fourthly, rodent ulcer and carcinoma of the skin.

With regard to the first of these, it is sufficient to remind the reader that we must not assume because a sore upon the skin is said to be, or may really have been, the result of a blow or a kick, that it is purely traumatic, for syphilitic ulcers often arise in this way. Malignant ulcers are rare and usually sufficiently obvious from the age of the patient, the pain they occasion, their tumourous margins, and blood-stained secretions. Moreover, they are with few exceptions confined to the neighbourhood of the orifices of the body, especially the lower lip, the urethra, the vulva, and the anus. Rodent ulcer, however, is very difficult to be sure of. Its locality, its slow and painless progress and its belonging to the latter half of life, usually serve to distinguish it from lupus, and unaccompanied with other signs of syphilis are the best characters for diagnosis from a tertiary ulcer.

Between lupus and syphilis the difficulty of diagnosis is occasionally extreme. Lupus, however, is rarely more than single, syphilis is usually multiple; both are commonly free from pain and itching, but in syphilis the colour tends from red to rusty brown, in lupus from red to violet blue; the scars of syphilis are depressed and pigmented, those of lupus hypertrophic and white; the edges of a lupous ulcer are beset with nodules, those of syphilis are either thin and smooth or indurated by chronic inflammation; lupus is in the majority of cases a disease of the face, syphilitic ulcers are quite as frequently on the limbs or trunk; lupus is a disease of the skin alone, syphilis affects the subjacent tissues also.

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* In the text this will inadvertently be found spelled Obermier.

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